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The Etiology of Milk Fever in Cattle.

By L. P. PUGH, B.Sc., F.R.C.V.S., Sevenoaks.

INTRODUCTION.

It has been pointed out in a previous communication (Pugh[2]), that in the course of "Milk Fever" in cattle, a syndrome develops which bears a very remarkable resemblance to that associated with intoxication by members of the botulinus group of bacteria, *i.e.*, by toxicogenic organisms whose poisons bring about bulbar paralysis. In this communication it was also pointed out that coma was not part of the botulinus syndrome, but must be due to some other underlying factor in the production of milk fever.

It is now proposed to deal more fully with this factor and to correlate it with conditions known to be associated with, or to predispose to, milk fever.

This factor is apparently an "acidosis" or, better, a "ketosis," by which term is implied a failure in metabolism characterised by excess of sugar in the blood, together with varying quantities of ketones, namely, aceto-acetic acid, B-oxybutyric acid and acetone. Ketosis is a well-known condition and is not peculiar to milk fever, although it has in this disease a chain of circumstances leading to its development, which circumstances are truly specific and entitle us to speak of milk fever as a separate and distinct clinical "disease."

It is convenient to consider this view of the etiology of milk fever in three sections:—

(1) The circumstances predisposing to the development of a ketosis, and precipitating its onset.
(2) The proofs of an existing ketosis.
(3) The mechanism of cure by inflation of the udder.

These sections will be followed by:—

(4) The relationship between the ketosis and the super-imposed bulbar syndrome.

All of these depend in some degree on an acceptance of current views on the subject of fat and carbohydrate metabolism, and these must therefore be set forth in some detail before other matters can be discussed.

FAT METABOLISM.

Fat taken in by the intestine has first to be saponified. This occurs in the intestine, and the altered fat is absorbed and then re-converted into fat in the villi of the intestinal mucosa. From there it passes to the liver, where it is burnt up with liberation of energy needed for various body functions. The final result of this burning is carbon dioxide and water.

If there is no immediate need for so much fat as is taken in, it passes through the liver unchanged (so far as is known) and is deposited in what are known as "fat depots," *e.g.*, the retro-peritoneal fatty tissues, etc. Here it remains until required. If a fat containing iodine is fed to an animal, that same type of fat will be found in the depots, or at least a closely-allied fat obviously derived from it.

We next suppose that a demand for fat arises, *e.g.*, from a falling off in food supply amounting to partial starvation. By some mechanism, of which we are entirely ignorant, the depot fat is liberated and passes to the liver where it appears in the form of globules of fat in the liver cells. Here it is broken down into carbon dioxide and water, and the energy liberated is used according to the body's needs. This process of breaking down is known as "desaturation," and is an orderly passage from more or less complex bodies until the simplest are reached. If it is incomplete, intermediate bodies appear in the blood, and these include those toxic substances found in the urine in diabetic coma and in milk fever, and these substances are known collectively as ketones.

This breaking down or burning up of fats is not accomplished without fuel, and the fuel is carbohydrate, of which the liver and muscles contain a store in the shape of glycogen. The relation of the quantity of fuel to the degree of fat metabolism is unknown, as are the details of the inter-action of the two processes; but that fat metabolism can only occur in the presence of some degree of carbohydrate metabolism is generally accepted.

In milk cattle, the liver has the duty of taking up depot fats, breaking them down into smaller and simpler molecules and then, instead of continuing the process until carbon dioxide and water are reached, of welding them up again into a form suitable for butter fat. We have not been able to find any publication bearing on the relation of butter fat to depot fat, or the conversion of one into the other, so that the sort of demand which butter fat formation makes on the liver is to some extent a matter of surmise. But the surmise is at least in line with what is already known of fat metabolism in general, and makes no new claim on the credence of the reader.

One factor of fat metabolism remains to be discussed, and that is the modification of the normal process which may be produced by an abnormal

demand for fat, *e.g.*, butter fat. Here, again, there is no strictly relevant publication to help us. We assume that the first result will be an excessive infiltration of the liver cells with fat derived from the depots. We know that this occurs in so simple a demand for fats as is occasioned by starvation. But there is some reason to suppose that this fatty infiltration can only be established at the expense of the normal glycogen infiltration, for in its presence the amount of glycogen is relatively low. If we look at the matter another way, we may suggest that normal fat metabolism requires the presence in each liver cell of a due proportion of glycogen (carbohydrate fuel) and a due amount of fat. So long as the fat does not increase so much as to displace this glycogen, metabolism is normal. When it does so increase (as seems to happen automatically if the demand arises) the fuel stock is found to have fallen too low to allow complete combustion, or in the case of butter fat, to allow of resynthesis of the immedaite products. There results the presence in the blood of aceto-acetic acid, B-oxybutyric acid, acetone, etc.

Circumstances Predisposing to Ketosis.

The circumstances here brought in evidence are generally accepted, and are those which have been verified by clinical observation by many persons and during many years.

1. *Milk fever was almost unknown before cattle were deliberately bred for milk production on the largest scale. This fact is clearly stated by Fleming*[1] *and many other authorities mention the point.*

It is obvious that a cow bred by careful selection to produce the greatest possible amount of milk must place a burden greater than the normal on that function of the liver which is concerned in converting depot fat into butter fat. It will to this extent be unfitted to bear the extra burden occasioned by the sudden transfer to the liver of a very large quantity of fat. Such a transfer seems to take place at just that period at which milk fever develops, and it is impossible to dissociate the two events, nor is it easy to escape the conclusion that it is the extra burden borne by the heavy milkers which determines in them the higher case incidence of milk fever. It seems possible that the actual fault is a displacement from the liver of carbohydrate fuel, but this particular explanation is not essential to the theory.

2. *Milk fever occurs at, about, or shortly after, parturition. It is rare before the completion of labour and occurs most frequently during the first forty-eight hours of the puerperium.*

It has been shown by Mottram[5] and others that there is a relatively sudden increase of fat in the liver at this time as, indeed, there must be if lactation is to commence normally. This increase disappears about the third day of the puerperium. Although neither Mottram's experiments nor those of any other author have been made on cattle (which are, of course too expensive for research work) it is reasonable to suppose that a similar process occurs in them. That milk fever should occur just when the burden on the liver is heaviest supports the theory that it is directly related to some metabolic abnormality, and the existence of a ketosis might almost have been suspected from this fact alone.

3. *Milk fever is favoured by excessive feeding just prior to parturition, and its incidence is definitely diminished where restriction of food during the last fourteen days of pregnancy is the rule.*

These two facts are both within the common knowledge of stock-breeders and herdsmen. They have never been explained by the infective or any other theory of milk fever. But they fall naturally into line with the metabolic theory here set forth as factors which either increase or diminish the work of the liver (or the displacement of carbohydrate) and are accordingly reflected in an increased or diminished incidence of the disease.

4. *Milk fever is rarely encountered in spare or thin cattle.*

This is a further clinical observation which is the converse of No. 1, and is only worth separate record because the observation is often made in this form.

5. *Milk fever very rarely occurs after abortions.*

We are bound to assume that fatty infiltration of the liver which characterises the close of pregnancy is undeveloped at an earlier stage. It is not impossible that the act of labour (even if an abortion) may do something to cause its appearance in some degree, but the scanty milk flow which follows abortion suggests that the liver cannot be functioning fully and makes it quite unlikely that it is ever so far burdened as to cause a ketosis. The failure of milk fever to appear after abortions thus accords well with the metabolic theory of its etiology.

6. *Milk fever rarely occurs after first labours or after difficult labour.*

In both cases there is (in the absence of milk fever) a relatively scanty flow of milk and this may be due to the remarkable state of turgescence (often seen in heifers) which the udder reaches in such cases. There is reason to suppose (as will presently be mentioned) that an empty udder causes in some way an increased flow of milk and that, on the contrary, the distension of a full udder inhibits the flow of milk. It is possible that the turgid udder of a difficult labour inhibits the flow of milk, thus lessens the demand on the liver and so minimises the chance of milk fever supervening. On the other hand it may be that we should seek to explain the fact in some other way. There is no doubt an increase of metabolism is occasioned by the energy expended in a difficult labour. This increase will probably affect first of all the combustion of muscle glycogen, but it may be that the demand is so great in some cases, or even in most cases, that the fat combustion in the liver is also accelerated.

It must be admitted that either explanation is largely hypothetical, but the metabolic theory of the etiology of milk fever is at least seen to offer some plausible method of approach to the rather puzzling fact that milk fever rarely occurs after difficult labour.

7. *Milk fever is precipitated by sudden complete emptying of the udder, and on the other hand is cured by inflation.*

Apart altogether from cases of milk fever we have a certain modicum of knowledge bearing on this point. During ordinary milking it is frequently observed that more milk is drawn off than could possibly have been in the udder at the commencement of the operation. In other words it seems probable that if we relieve the distension of the udder a stimulus is provided which results in further milk formation. The udder fills up according to the needs of the calf, and the index to these needs is the extent to which the supply already in the udder has been depleted. It is also known that distension of the udder inhibits the flow of milk, even in cows which have not milk fever. Such distension is occasionally resorted to for a variety of purposes which are not at the moment in question.

What is true in other circumstances is probably true in this matter of milk fever. Complete emptying of the udder acts indirectly as a stimulus to the metabolic activities of the liver, since it calls for fresh transferring of depot into butter fat. Equally the distension of the udder (whether by air or fluid) slows down these processes. By so doing it diminishes or abolishes the formation of ketones and allows of recovery. The metabolic theory affords an explanation of the relationship which is at least possible and even probable, so that it may well claim to stand until a better is forthcoming.

There are other and minor circumstances which are recognised to be associated with milk fever, but those already mentioned are admitted to be the most important by the majority of authorities and serve sufficiently to illustrate the thesis that the metabolic theory harmonises a number of facts which have hitherto appeared discordant.

The Signs of Ketosis.

Although coma is typical of advanced ketosis, yet it is not pathognomonic in that coma can occur in other conditions.

It is essential therefore that these ketones should be demonstrable in all cases of milk fever, and the most certain and reliable method is that of urine examination. Ketones are excreted very largely by the kidneys.

There are a number of tests that can be applied, but the one that commends itself by its simplicity is that introduced by Rothera[4].

To 5—10 c.c.of urine or distillate in a test tube add a little solid ammonium sulphate, 2—3 drops of a freshly-prepared 5 per cent. solution of sodium nitroprusside and 1—2 c.c. of concentrated ammonium hydroxide. The development of a permanganate colour indicates the presence of acetone and acetoacetic acid.

With practice it is possible to gauge roughly the amount of ketones present by the rapidity with which the colour reaction is developed and also by the intensity of the colour. In urine obtained from comatose milk fever cases, the reaction can be so intense as to become "black" almost immediately. On the other hand, if a cow is detected in the very early stages, the permanganate colour is developed slowly and does not become black.

(N.B.—Acetone is said to be found in small quantities in the urine during the earlier part of the puerperium.)

But quantitative analysis has shown that acetoacetic acid is in far greater amounts than can be explained on physiological grounds.

It has been mentioned that hyperglycæmia is associated with ketosis. Hyperglycæmia can occur without ketosis. However, a number of quantitative blood sugar tests have been carried out and these have shown that the blood sugar is considerably higher during milk fever than after recovery.

Ketosis and the Bulbar Symptoms of Milk Fever.

The theory already set forth has dealt with only the etiology of milk fever. Something must now be said as to its bearing on the symptoms.

As already noted, these closely resemble those produced by organisms of the botulinus group. The degree of resemblance is well suggested by a recent communication by Theiler, Green and Du Toit[3], in which lamziekte is stated to be brought about by a bacterium "reminiscent of, but not identical with, *B. botulinus*." So, also, the symptoms of milk fever are reminiscent of, but not absolutely identical with, those of botulism.

The symptoms of ketosis other than the coma are of a very indefinite nature if we are to judge from textbooks on diabetes, etc., in which they are customarily set forth. On the other hand, those of milk fever are quite distinct and are due to a paralysis of the bulbo-sacral nervous system. Yet they clear up after the use of that same remedy, namely, inflation, which stops the ketosis. It seems clear that they depend in some very direct manner on the ketosis, and this deduction is none the less valid even if we suppose them to be of bacterial origin.

Although the relationship appears at first sight somewhat difficult of explanation, we have analogous happenings in the cases of lamziekte in South Africa and parabotulism in Australia. Both of these are well-known to occur with particular frequency at or about the time of parturition. It is not disputed that they are bacterial diseases, so that we are forced to conclude that parturition in some way favours their development. It is possible that the normal metabolic changes of parturition hinder the body defence against toxins of the botulinus group. It is this connection, it is pertinent to note, that these changes affect most seriously precisely that organ, the liver, which is first responsible for dealing with toxins of alimentary origin.

But there is another possibility which must also be considered. In milk fever there is considerable intestinal stasis. We have reason to suppose that this alone, however brought about, may suffice to

determine the entrance into the blood of toxins of intestinal origin. For example, sub-acute lead poisoning is not usually credited with producing bulbar or botulinus-like symptoms. Yet, in cattle, these symptoms do supervene if lead poisoning is sufficiently intense and prolonged, and the writer has encountered such cases and has had difficulty in arriving at a definite diagnosis until the source of lead was brought to light and eliminated.

On the evidence produced we are entitled to assume that the factors which determine the entrance into the blood of botulinus-like toxins may be of almost as great importance as the toxins themselves. Nor do we lack evidence to suggest that amongst these factors parturition has a definite place, although we may be unable to provide a convincing explanation of its mode of action.

One could not, without unduly prolonging the paper, discuss any further the relationship of the symptoms of milk fever to the metabolic theory of its etiology, but it may be noted that some of the less often appreciated results of inflation support the view that events move somewhat in the order set out below :—

1. Ketosis.
2. Reduced resistance to toxins.
3. Admission to the circulation of botulinus-like toxins.
4. The symptoms of milk fever other than coma.
5. Inflation.
6. Cessation of ketone formation and commenced elimination of those already formed.
7. Restoration of power to deal with toxins (possibly by return of peristalsis).
8. Neutralisation of toxins.
9. Recovery.

The metabolic theory of milk fever has now been set forth as shortly as possible, and the following tentative conclusions are suggested as a working basis for further research :—

(*a*) Milk fever is predisposed to or precipitated by factors which hinder fat metabolism in such a way that ketones are produced and circulate in the blood.

(*b*) It is alleviated or cured by factors which diminish the call for butter fat, in which category is inflation of the udder.

(*c*) If the particularly urgent demand created in the first few days of the puerperium by the inception of lactation can be safely passed, milk fever is unlikely to occur.

(*d*) The majority of the symptoms of milk fever are caused by botulinus-like toxins derived from the alimentary canal.

(*e*) Death may be caused by the ketosis or by the toxins, or, as is probably more usual, by both combined.

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Enzootic Jaundice (Yellows) in Dogs.

By C. C. OKELL and T. DALLING, Wellcome Physiological Research Laboratories, Beckenham, Kent; and L. P. PUGH, Sevenoaks.

We have recently had the opportunity of investigating infectious jaundice in dogs. The cases so far have come from an area in each of three counties, Kent, Herts, and Surrey. The condition is similar to Weil's disease in human beings, the course and *post-mortem* appearance being alike. Careful search was made for piroplasms, but none were found in any of the animals. We have found a leptospira present in various tissues of several affected dogs and in rats found in close association with the infected kennels. This leptospira when injected into guinea-pigs and dogs, either as culture or a liver emulsion from naturally infected dogs, has produced the disease with typical symptoms and *post-mortem* appearances. Fortunately we had at our disposal supplies of anti-leptospira serum which one of us (C. C. O.) had made by immunising horses with rat leptospira obtained from Scottish mines in which cases of human jaundice (Weil's disease) had occurred. If guinea-pigs be protected with this serum they withstand doses of culture or infected tissue fatal to unprotected guinea-pigs. The serum of dogs which have recovered from the disease appears to protect guinea-pigs against an otherwise fatal dose of culture or infected tissue. Dogs infected with the rat strain of leptospira develop a clinical and *post-mortem* picture indistinguishable from that of the natural disease.

The anti-leptospira serum has been used prophylactically and therapeutically. Few results are yet available, but all seem to be favourable. In one infected kennel seven dogs were given serum, none developed the disease; of five not given serum, two have died of the disease. A full account of the investigation will be published when the work has further advanced. We believe the disease is widespread in the country, but no reliable data are available. We would be very grateful to veterinary surgeons for notes of any cases observed by them. We do not at present know of any human cases of the disease in association with these canine outbreaks, but enquiries are being made.

Conclusions.—(1) Enzootic jaundice (yellows) in dogs has been investigated. The disease is apparently caused by leptospira icterohæmorrhagiæ.

(2) Anti-leptospira serum apparently protects against the disease. It has been tried curatively in a few cases with promising results.

(3) As leptospira icterohæmorrhagiæ is spread by rats, energetic anti-rat measures are advisable in all infected kennels.

Royal Army Veterinary Corps.

LONDON GAZETTE—WAR OFFICE—REGULAR FORCES.

November 18—Captain (Bt. Major), A. F. Castle, F.R.C.V.S., is appointed D.A.D.V.S., of a Division. (November 17).

Anærobic Infections in Animals.

By S. H. Gaiger, F.R.C.V.S.,
Director, Animal Diseases Research Association Laboratory.

(*Continued.*)

If long bacterial filaments are present the case is one of Vibrion septique infection. These filaments may be found as soon as death occurs, but are more certainly found if the body is left unopened for an hour or two. If bacilli are present, and only lying in ones and twos, or if not present at all, the case is probably a B. chauvœi infection. Only one with much experience of both organisms in the laboratory would venture to distinguish between the two organisms in smears from the muscle lesion. The B. chauvœi navicular form is in the form of a spindle, whereas that of Vibrion septique is much more of an oval. A guinea-pig inoculated subcutaneously with liquid from the lesion dies much more rapidly from Vibrion septique than B. chauvœi. From the former it will be dead and cold inside twenty-four hours, whereas from the latter it may not die till forty-eight hours, though, of course, much depends upon the dose given. The Vibrion septique is much more invasive of the system than B. chauvœi. The intestines of the guinea-pig dead from a Vibrion septique infection are highly inflamed. This is not so with B. chauvœi. In cultural characters the two organisms are unmistakeable. B. chauvœi will not grow in 2 per cent. glucose, 2 per cent. agar medium, whereas Vibrion septique grows profusely in twenty-four hours, breaks up the entire medium in a shake culture, and blows the cottor plug out of the tube. In all media in the presence of fermentable carbohydrate Vibrion septique grows abundantly in twenty-four hours with formation of numbers of gas bubbles. The only medium in which B. chauvœi might be said to grow abundantly is liver broth. Surface colonies of B. chauvœi are like clear water droplets, whereas those of Vibrion septique are flattened, more opaque, and there is a tendency for the growth to appear as a film spreading from the water of condensation in the tube. Deep colonies of Vibrion septique have a dense centre with a fluffy halo of filaments, easily seen with a hand lens, whereas those of B. chauvœi have a solid lenticular centre with a burst of filaments at one point. The great feature of cultural difference between the two organisms, however, is that unless very strict anærobic conditions are maintained for B. chauvœi no growth will result (except in the case of liver extract media), whereas considerable liberty may be taken with Vibrion septique in this respect.

I shall feel I have made my point if I have convinced you that the clinical entity called blackquarter is not a bacteriological entity, and that the two chief organisms which can set up this lesion, the B. chauvœi and the Vibrion septique, are quite distinct organisms, and readily distinguishable.

Immunisation.—You are all aware of the standard methods of vaccination against B. chauvœi: the methods (double and single inoculation) with attenuated virus in the form of muscle powder; the method of using anti-serum along with culture; and the method of using a spore vaccine upon threads, etc. The method to which experimental attention has been devoted more of late is immunisation with aggressins (sterilised œdematous fluid from the lesions) and culture filtrates, which contain a soluble toxin produced during the multiplication stage of the organism in the medium. Foth[7] in 1909, Nitta[10] in 1918, Eichhorn[11] in 1918, and Kelser[12] in 1918, all showed that cultures possess immunising substances in solution. Haslam and Lumb[13] in 1919) found that B. chauvœi filtrates as issued by four different laboratories in America were non-toxic to guinea-pigs, and possessed marked immunising properties in calves. They consider their experiments indicate that the immunising power of B. chauvœi culture filtrate is not dependent upon toxicity of the filtrate. In their experiments each calf had 5 c.c. filtrate, and after at least two weeks was tested with 10 c.c. culture. Of fourteen calves inoculated only two were susceptible. Ten controls all died of blackquarter. Hart[14] in 1919 describes unfavourable results in two calves, 3 months and 1 month respectively, which were inoculated with culture filtrate, and later developed natural blackquarter. Ward[15] in 1919 publishes experiments to show the protective value of aggressin. Welch and Marsh[16] in 1921 record four animals inoculated with 3 c.c. commercial aggressin, which later withstood a test dose of virus. Two controls died.

The Vibrion Septique (Pasteur, 1877.)

Usually termed in veterinary literature the bacillus of malignant œdema. It is also known in literature as the Bacillus of Ghon and Sachs, 1903. In morphology and cultural characters this organism resembles the B. chauvœi, except for the differences which have already been given. These differences may be briefly recapitulated. The Vibrion septique forms filaments unevenly segmented on the peritoneum of animals which die from infection with it. Its navicular forms in the lesion and in culture are more ovals than spindles. It is less strictly anærobic, grows much more rapidly in meat media, producing considerable cloudiness and gas formation in eighteen hours with a bright red colour in the meat, whereas B. chauvœi only shows commencing growth in the same period. It grows readily in 2 per cent. glucose 2 per cent. agar medium, with much breaking up of the medium, whereas B. chauvœi does not grow. In surface culture on blood agar there is a tendency to a film-like growth, individual colonies are flattened and somewhat opaque, contrasting with the individual, raised, clear-water-droplet character of B. chauvœi colonies. Deep colonies have a dense centre with a fluffy halo as compared with the lenticular colony and filamentous outburst of B. chauvœi. It ferments salicin and does not ferment saccharose—this being

vice versa for B. chauvœi. It grows readily in litmus milk, whereas B. chauvœi does not. The Vibrion septique, in the bulk of the literature prior to 1918, is stated to be a proteolytic or putrefactive organism due to work being done with cultures contaminated with B. sporogenes. This is now known to be wrong. The Vibrion septique is not a proteolytic organism, and does not produce an unpleasant smell in pure culture. A perusal of literature shows that Vibrion septique has been frequently isolated and identified as B. chauvœi from animal diseases.

Distribution in Nature. Carl[17] inquired into the distribution of Vibrion septique and concluded that though widely distributed in soils it is not ubiquitous. Cornevin found it rare in forest earth. Jensen[20] found it rare in earth around Copenhagen. Feser infected 7 out of 30 guinea-pigs with filthy earth. The writer has examined cultivated soils from several different counties in this country, and found Vibrion septique in all. This organism is a constant inhabitant of the alimentary canal of herbivora. On the death of the host it accompanies such putrefactive anærobes as B. sporogenes when they invade the carcase.

Pathogenicity.—Heller[5] examined a number of strains from various sources in America, and records that thirteen were from cattle, eight from sheep, one from a horse, and two from pigs. There are many records of Vibrion septique infection in cattle following wounds and parturition. Many cases are recorded in horses following wounds. At least some cases of "puerperal blackquarter" in cattle have been shewn to be Vibrion septique infection, as have some cases of muscle blackquarter. Lembeken[18] records fifty out of six hundred freshly-shorn sheep as having died of "malignant œdema." Likewise docking and castration in sheep have been reported as followed by the disease. Meyer[19] records an outbreak of blackquarter in pigs, and shows it to have been a Vibrion septique infection, and there is ample further evidence of like infections. Jensen[20] describes his bradsot bacillus in sheep in Iceland leaving no room for doubt that this organism is the Vibrion septique. The writer[21] has shewn his braxy bacillus to be identical with the Vibrion septique, and doubtless "black disease" of sheep in Australia will prove to be due to the same organism. It may be taken as generally the rule that anærobic infections in sheep are due to the Vibrion septique, and that the lesion arises in the alimentary canal, and also that anærobic infections in cattle are usually due to B. chauvœi and the lesion situated in the muscles, but this is by no means constantly so as has already been shewn. There are records (Dinwiddie[22]) of pig infections with Vibrion septique where no muscle lesions or wounds were found, but with alimentary lesions comparable to those found in braxy in sheep. Upon artificial inoculation subcutaneously the Vibrion septique produces extensive hæmorrhagic œdema subcutaneously and in the underlying muscles, and a considerable amount of gas. The hair over the lesion slips out very easily. The peritoneal surfaces are very moist, the intestines contain a considerable amount of gas, and there is constantly present excessive injection of the peritoneal serous membrane, and of the intestines. The suprarenals are hæmorrhagic. Some strains on subcutaneous inoculation produce marked gastritis. Cultures from the heart blood yield the organism in the great majority of cases. Upon subcutaneous thigh inoculation in sheep the underlying muscles show a lesion indistinguishable from blackquarter. This lesion at times may be as far removed from the seat of inoculation as the muscles in the sternal region, leaving the thigh muscles almost unaffected. Intravenous inoculation into the jugular vein of sheep gives rise to a blackquarter lesion in any part of the skeletal muscles or to an abdominal lesion. Jensen[20] fed 1,545 sheep with pure culture, and five died of typical bradsot, i.e., with the abomasum as the seat of primary location of the bacillus. Dogs and cats only show a local lesion on inoculation with the Vibrion septique. The rabbit is relatively insusceptible. Whereas ·005 c.c. of twenty-four hours pure culture or less on subcutaneous inoculation may be fatal in twenty-four hours to a guinea-pig, 1 c.c. may have no effect upon rabbits, which may survive 2 c.c.

Immunity.—Like the B. chauvœi, the Vibrion septique produces a soluble toxin during its multiplication stage which diminishes rapidly in quantity on further incubation. The culture filtrate upon inoculation into animals has been shewn by several observers to stimulate the production of antibodies. The writer has made use of this fact in preventive inoculation of sheep against braxy. Jensen's method[20] against bradsot is to use a sero-vaccine.

The Vibrion Septique as the cause of Braxy in Sheep.—The writer has made an exhaustive study of braxy in sheep, in which the losses in this country are anything up to 35 per cent. of sheep in the braxy districts in their first winter of life (hoggs). By residing in the braxy districts, with a well-equipped field laboratory, live cases of braxy were obtained and killed when near the point of death. In all cases a constant stomach lesion was found. Histological preparations from these lesions showed conclusively (lantern slides will be exhibited) that the lesion is an inflammatory one; the bacilli can be seen in pure culture in the wall, which is enormously thickened with inflammatory œdema, and large numbers of leucocytes are seen to have migrated from the distended blood vessels and to be attacking the bacilli. In many of the sections localised hæmorrhages may be seen in the mucosa. The surface of the mucosa is seen to be undergoing necrosis. Under the most stringent precautions, and with a technique carefully prepared for beforehand, pure cultures were obtained of the Vibrion septique from the wall of the affected unopened stomach by penetration through the peritoneal coat from the peritoneal fluid, and from other organs and fluids in the body, including the heart blood. This organism was invariably present and in pure culture. An extensive study of the organism was made as to its morphology, cultural characters, pathogenicity, serological and immuno-

logical reactions, leaving no room for doubt that braxy is a pure infection with the Vibrion septique. The organism proved to be invariably fatal to sheep upon subcutaneous and intravenous inoculation and produced gastritis and enteritis in addition to the local "blackquarter" lesion. From such inoculated cases in sheep the Vibrion septique was again obtained in culture.

The principal exciting cause of braxy was shewn to be the filling of the rumen with frosted food, resulting in a chilling of the abomasum, which lies in close contact with it. Any factor which lowers the tone of the fourth stomach may allow the Vibrion septique to penetrate. There is no need for me further to detail the work of the braxy investigation, since it is accessible to all in the *Jour. of Comp. Path. and Ther.* for 1922. In that article the question of immunisation is dealt with, giving the preliminary work done on field immunisation of hoggs with culture filtrate. Since the publication further inoculations done upon many thousands of hoggs in Scotland, England and Ireland show that inoculation reduces the death rate to one quarter what it is in the uninoculated. Further, I have been in communication with representatives of the Bureau of Animal Industry in America, and at their request have sent vaccine. While writing this paper I have received a report that they are getting very encouraging results upon a small scale, and are commencing to make the filtrate from their own organism obtained from sheep in America. I have examined this organism and find it identical with mine. The published results have also created interest in Australia, to which I have sent cultures by request, and where endeavours are to be made to protect sheep against "black disease" by means of the filtrate, since "black disease" in sheep is very probably identical etiologically with bradsot and braxy. The filtrate is also being issued by the Department of Agriculture and Technical Instruction in the Irish Free State, and by the Department of Agriculture in Northern Ireland—both departments having sent representatives over to see the methods at first hand.

The writer's results have been accepted as conclusive by the scientific world, and as shewn above have led to world-wide action on sunilar lines against the disease.

The Vibrion septique, from whatever source it has been isolated in animals and man, is accepted by bacteriologists in every country in the world as a non-proteolytic or non-putrefactive organism. Some of the most skilled bacteriologists in every country have of recent years been engaged in work upon anærobes, and are unanimous in the opinion that the pre-war conception of Vibrion septique as a putrefactive organism was wrong, and the error was due to working with impure cultures. The cultures I have obtained from sheep were pure in primary culture, and this purity has been confirmed by leading bacteriologists in several countries. All my strains are non-proteolytic.

I lay emphasis on these points because a criticism has recently been published in English in a German journal by McGowan[23] and reprints of this article have been issued in this country by the Rowett Institute for Research in Animal Nutrition. This article contains no new research work. It is merely a criticism of the work of others upon braxy, followed by a re-statement of McGowan's work upon one sheep obtained in 1912 as soon as it was dead, and from which McGowan concludes that braxy is hæmorrhagic septicæmia.

The tone of McGowan's criticism does not require more than mere mention. His points can be disposed of under a few headings.

The Stomach Lesion.—I have recorded detailed observations upon thirty cases found and brought alive to a field laboratory, and in every case a distinct, characteristic, and unmistakeable lesion has been found to exist. Besides these recorded cases I have opened numbers of dead braxy sheep and found the same lesion though masked by putrefactive changes. McGowan claims that the stomach lesion is inconstant. In none of his writings does he give any reason to believe he has dealt with more than one *live* case of supposed braxy. My findings are in accord with the views held by intelligent shepherds who are in the habit of opening braxy sheep. Hogg[24] for example, in 1829, says he "has dissected several hundreds that have died, and . . . there has not been above one case in a hundred where it was not evident that the fourth stomach was and had been the first and principal part affected."

I have shewn that the nature of the lesion in all my cases is an inflammation due to an invasion of the tissues by the Vibrion septique. There is congestion of the veins with or without localised hæmorrhages, an inflammatory exudate into the tissues and leucocyte migration to attack the invading bacilli. Hæmorrhage is only to be seen in the mucosa, but it is localised and is a result of the invasion of the bacilli, and not the cause of it, as McGowan states. In some cases no sign of hæmorrhages can be seen in the lesion. Neither Hamilton nor McGowan record any detailed histological study, so it may be presumed they designated the lesion a hæmorrhage from naked-eye examination. McGowan's view, that the lesion is an infarct with leucocytes migrating to remove it, is not in any way whatever borne out by what one finds on histological study. This I will show you in lantern slides.

Putrefactive Organisms.—My technique has shewn braxy to be due to a pure invasion of the Vibrion septique from the fourth stomach. McGowan in his recent article reiterates the view that Jensen's bradsot organism and my braxy organism are putrefactive bacteria, and propounds his infarct theory to show how such bacteria may invade the body during life. My braxy organism has been shewn identical with that found by Jensen in bradsot. It follows then that McGowan looks upon the Vibrion septique as a putrefactive organism. He probably stands alone in the scientific world in this belief, in view of the world's

work upon this organism since 1918. McGowan gives records of one case dealt with as soon as it died in 1912. In 1915 he records of this case: "I had the good fortune to obtain one of the cases as soon as it died, and consequently . . . the carcase was entirely fresh." Nine years later (1921) he lays much stress upon this fact, and in 1923 he again lays much stress upon it. Realising the value he attaches to this point it is interesting to note his views in an article entitled "Braxy in Hares," 1921. McGowan[23] dealt with hares found dead of braxy and sent to him. From large hares he obtained "a pure extensive uncontaminated culture of an organism of the hæmorrhagic septicæmia group." From small hares he obtained no growth, although they "must have been dead in some instances for days before they were examined." Then McGowan points out "the necessity of examining conscientiously every specimen, even a presumably putrefied carcase, if nothing better can be had." Since 1915 McGowan has received many dead hares. In all specimens "the same organism was obtained in pure extensive culture from the heart blood, spleen, and liver," and "in one case from the small intestine." It is very difficult to know what to make of such cultural results. My cases of braxy were obtained alive; some were bled to death, others died and were bled when the heart had ceased to beat. There was no difference in the bacterial findings. McGowan allowed his case to die, and gives his view that cutting the throat allows the heart blood to become contaminated. In my cases where cultures were made from the heart the blood from the left ventricle was used and yielded pure cultures of the Vibrion septique. It is not easy to follow the reasoning that cutting the throat can contaminate such blood, nor how it can contaminate it with a *pure* culture of anything, let alone an anærobe. It is stated by McGowan that I have generalised from twenty cases. This should read thirty. Even if one were to claim that it is unsafe to generalise from such a large number, all of which were identical, it is at least safer than generalising, as McGowan has done, from one case.

McGowan's 1912 sheep outbreak occurred in August, which is not the braxy season. This fact, coupled with his findings, justify one in concluding that it was not a case of braxy which he dealt with. This is further borne out by the fact that the case originated on its own land and lived long enough after it was found to be brought to the laboratory of a "large institution." It is unfortunate that McGowan says nothing regarding the clinical symptoms in the 1912 case he dealt with. His organism did not produce the post-mortem lesions of braxy, nor did those lesions have more than a passing resemblance to hæmorrhagic septicæmia, of which I have had a wide experience abroad.

Finally, McGowan claims that his organism causes other diseases as well as braxy, namely, trembling in sheep, head grit in sheep, lamb dysentery, and bracken poisoning in cattle. I would suggest that his organism from hares and from this variety of sheep and cattle diseases is the Bacillus coli. In a private communication, Dr. McGowan informed me that his cultures from the 1912 sheep have long since died out, so his 1912 findings cannot now be checked; suffice it to add that in none of my killed or died affected sheep was anything met with comparable with his findings. No ærobic growths in culture were obtained in my cases from any organ or fluid in the body.

The fact that there is a disease of sheep in several countries in the world caused by a pure invasion of the Vibrion septique is not without interest to the human pathologist as well as the animal pathologist. The soil is the common source of infection for animals and the war wounds of man, and it is not surprising to find that the organisms from man and animals are identical. I have pointed out how the war found our knowledge of soil anærobes to be lacking, and much time was expended during the course of the war in acquiring the necessary knowledge; indeed, it was not till the war was ending that anærobe antisera became available. Had animal diseases been studied prior to the war to the extent they ought to have been studied, at least the Vibrion septique would have been better known to science in pure culture, and mankind would have benefitted by the better understanding of animal pathology.

Some years ago I received from the late Mr. Wallis Hoare a number of specimens for examination from cases of so-called "gloss anthrax" in bovines. These specimens consisted of smears of fluid from the œdematous swellings, and one or two ears from such cases. The specimens sent were unsuitable for cultural study, but they were undoubtedly infections with one of the pathogenic anærobes, and judging by morphology alone, probably Vibrion septique infections. I would welcome the opportunity of getting into touch with practitioners who meet with such cases in their practice.

The Vibrion Septique in Lamb Dysentery.—I have lately found the Vibrion septique in a situation where it almost certainly exercises a pathogenic rôle, and that is in the ulcers of dysentery in lambs, where it is present along with Bacillus coli, Bacillus welchii, and gram-positive cocci. In this disease the Bacillus coli seems to set up a catarrhal condition of the intestines, predisposing to the invasion of the intestinal wall with the two mentioned pathogenic anærobes. The ulcers in lamb dysentery become analogous to the war wounds of man, and from their situation—the delicate bowel of the new-born lamb—rapidly bring about a fatal termination, by the production of toxins in the ulcers, or by multiplication and toxin production in the organs and fluids of the body after penetration from the ulcers, along with the Bacillus coli.

The Bacillus Welchii (Migula, 1900).

This organism, although described as long ago as 1892 by Welch and Nuttall under the name B. ærogenes capsulatus, really came into prominence in connection with gas gangrene in war wounds. It is also commonly

known as Bacillus perfringens. Descriptions of this organism have suffered from work done with impure cultures. Observations prior to Simmonds' Monograph of the Rockefeller Institute, 1915, are mainly unreliable for this reason.

Morphology.—It is a strongly gram-positive, non-motile, sporulating, short and thick rod, with squarish ends, measuring from 4 to 8 microns in length to 1 to 1·5 microns in breadth, found singly or in twos and threes and short chains. It sporulates freely in the intestinal contents of animals, but its spores are seldom seen in artificial culture. Spores may be found in surface colonies on blood agar or in media to which 2 per cent. casein digest has been added, or in alkaline egg medium after three weeks' incubation. The spores are large, oval, and situated centrally or subterminally.

Cultural characters.—The B. welchii grows very rapidly under anærobic conditions, producing a very large amount of gas. It is the only non-motile anærobic bacillus which produces the so-called "stormy fermentation" in milk. On the surface of potato-agar and blood-agar colonies are formed, which are at first clear and later opalescent. They have a bluish-grey shining appearance, and may be one or two millimetres in diameter. The colonies are hæmolytic. One strain received from a human source (gas gangrene) gives colonies of viscid consistency which when hooked on a needle, pull out like elastic. This strain turns liquid media into the consistency of syrup. No strains of B. welchii from animal sources isolated and studied in the A.D.R.A. laboratory present this particular feature.

Deep colonies in 0·1 per cent. glucose agar 1 per cent. show a solid granular centre, with usually a few filaments at one end or the side of the colony. The outline of the colonies is not hard and sharp as with B. chauvœi. Their shape is oval or round. The medium becomes entirely broken up by gas formation.

The B. welchii is not very strictly anærobic, indeed Caulfield[25] records that he frequently found it grow ærobically on the surface of agar, but the bacilli were longer and finer and without virulence.

In meat media this anærobe produces very rapidly a dense cloudiness with tremendous evolution of gas, with a sour odour, and a pink colour in the meat. In litmus milk medium, acid and clot are formed, the clot being riddled by the production of gas in enormous quantity. Wolf and Harris[26] showed that as much as 2,820 c.c. of gas is produced from 1,000 c.c. of milk in twenty-four hours, and that a pressure of gas of two atmospheres is produced in a few hours. The Bacillus welchii ferments glucose, lactose, saccharose, starch, glycerin, lævulose, galactose, maltose. Gelatin becomes softened after three days' incubation. It can only be said to be mildly proteolytic. The presence of utilizable carbohydrate discourages spore formation. In liquid culture media this organism produces a toxin during the initial stage of rapid multiplication, but prolonged incubation greatly diminishes the amount of toxin. If the culture is filtered after eighteen to twenty-four hours incubation a relativel y stable toxin is obtainable. The toxicity of a forty-eight hour culture is almost negligible.

Distribution in Nature.—The B. welchii is widely distributed in nature. It is probably to be found in all cultivated soils. It is present in the intestinal canal of healthy animals of probably all species. Koser[27] records that he found a commercial bread starter to contain organisms of this type, but of a low grade of virulence. In the A.D.R.A. laboratory it has been found in the vaginal passage of ewes, and upon their udders and teats as well as in many soil samples.

Pathogenicity.—There is a noteworthy absence of records of serious invasion of the living tissue of animals. Strains of animal origin studied by Kendall, Day, and Walker[28], were, one from the fæces of a dog, and one form the fæces of a horse. One of Hall's six strains was from the tissues of a cow dead of "redwater," but there is nothing to show tissue invasion during life. Von Hibler[30] isolated it from the tissues of a pig thought to have died of swine fever. The Medical Research Committee[31] report that B. welchii apparently exists as a saprophyte in many wounds without etiologic importance. The environment must be of a nature which encourages the production of exotoxin before this bacillus exerts a pathogenic effect. Sacquepée[32] considers that, though the bacillus is important in war wounds, its pathogenicity only comes into play as a secondary invader. Caulfield[25] found that the pathogenicity of other micro-organisms is increased when present in a host experimentally infected with B. welchii. Its presence in lambs infected with dysentery will be recorded presently. Upon experimental inoculation most strains of B. welchii are highly pathogenic organisms. On subcutaneous inoculation of guinea-pigs it produces a slightly blood-tinged emphysematous œdema, and the animal dies within twenty-four hours. Sublethal doses produce intoxication and a local slough which ultimately heals. Recovery of a guinea-pig does not prevent a repetition of the illness and even death upon re-inoculation, but after repeated inoculation with increasing doses the animal becomes immune to further inoculations (Robertson[33]), (Hall[29]). B. welchii can be recovered from the heart blood of a guinea-pig dead after inoculation, but not with the same certainty that the Vibrion septique can. The invasive powers of B. welchii seem to be considerably less than Vibrion septique. Rabbits are less susceptible than guinea-pigs, the same lethal guinea-pig dose producing intoxication and a local lesion, from which recovery occurs. The pigeon is very susceptible to intramuscular inoculation. Caulfield[25] found that ·01 c.c. was fatal in eight hours. The bacillus, minus its toxin, has been found non-pathogenic.

The Bacillus welchii in Lamb Dysentery.—It behoves veterinarians to be on the look-out for this bacillus in animal diseases, since the absence of records of infection may be in part due to lack of recognition. It is improbable that there can be any

great number of cases, if any, of pure infection, but the possibility of these as well as of mixed infections should be borne in mind. In wound discharges the presence of a relatively short and thick strongly gram-positive organism not shewing spores should make one think of B. welchii and its possible pathogenic significance. Such discharge might with advantage be forwarded to a laboratory for examination as to its bacterial flora simply in the interests of science.

In my laboratory the B. welchii has been found present in the ulcers in lamb dysentery, a disease referred to under the description of Vibrion septique infections. Its precise significance in this disease is not clear at present. Were it confined to the ulcers it might be relegated to a minor significance, but in some cases it is found invading the organs of the body in company with B. coli, Vibrion septique, and gram-positive cocci. The view which at present seems justified is that B. welchii has some pathogenic significance as a secondary invader of the bowels, weakened by a virulent type of B. coli. The ulcer in the young lamb's intestine seems to be analogous to the war wound of the human being, in that it is the seat of multiplication of a number of fæcal organisms. That B. welchii is not of primary etiologic importance in this disease is indicated by the fact that an antiserum for this organism does not seem to have any influence in stopping the onset of a natural attack.

Presence of the B. welchii in other Diseases of Sheep.—In the A.D.R.A. laboratory the B. welchii has been found at times associated with the sheep diseases known as trembling and scrapie. It has been found in the brain substance of both of these diseases, which are of the nervous type, and both in pure and mixed culture. The significance of the presence of these organisms in the brain tissue in such diseases is being studied.

Immunity.—Several observers have placed it on record that an antiserum to B. welchii of high titre can be produced in a relatively short time. The Medical Research Committee found that a serum with a titre of 1,000 units can be produced from the average horse in about eight weeks. Christiansen records that normal horse serum protects guinea-pigs against infection with B. welchii.

BACILLUS ŒDEMATIENS (Weinberg and Séguin, 1915).

This sporulating anærobe was isolated by Weinberg and Séguin from cases of gas gangrene in man. It was discovered by Novy in 1894.

Morphology.—This bacillus measuring 3 by 1 microns is grampositive, sluggishly motile when examined under anærobic conditions, relatively thick like B. welchii, though usually longer, with ends more rounded, and sporulating more readily. The oval spores are large and subterminal giving a tennis-racquet appearance, or central. It is found singly or in pairs, but is occasionally found in short chains and filaments. It does not form filaments on the peritoneum.

Cultural Characters.—The B. œdematiens grows readily under anærobic conditions. On the surface of blood agar young colonies resemble those of B. welchii, and become flatter with further incubation running into each other to form a film. Deep colonies in agar are fluffy and like snowflakes; more fluffy and filamentous than those of Vibrion septique, from which they can readily be distinguished. In meat medium there is cloudiness and gas formation and the meat turns a faint pink. There is no action on milk, or a delayed formation of acid and clot after prolonged incubation. Gelatin is liquefied. It is a non-proteolytic organism resembling Vibrion septique and B. chauvœi closely in its biological characters. It ferments only glucose, lævulose, xylose, maltose, and starch. As with the other anærobes described, a filterable toxin can be obtained from young cultures.

Distribution in Nature.—This bacillus can be shewn experimentally to exist in cultivated soils.

Pathogenicity.—The B.œdematiens has been recorded from animals upon several occasions. It was probably this bacillus which Kerry[34] found in Germany, in 1894, in the muscles of a cow supposed to have died of blackquarter. Von Hibler[30] reported it in 1908 in Germany in a wild boar dead from supposed blackquarter. Hall[29] in America, in 1920, found it in a fatal wound infection in a horse, and apparently pure. Weinberg and Séguin[35] record a case in a horse which was being used for the preparation of antivenin. The B. œdematiens was present along wth the B. histolyticus, but the former was identified by its morphology alone, since it could not be obtained in pure culture. Heller[5] records three fatal cases in horses of what was probably œdematiens. She examined material obtained indirectly from horses being used in "an immunity experiment." There was a subcutaneous gelatinous œdema without gas formation.

The B. œdematiens is highly pathogenic for the guinea-pig—a fraction of a cubic centimetre of culture causing death on subcutaneous inoculation in twenty-four hours. Death may occur rapidly from intoxication before the bacillus has time to invade the system, but usually cultures from the heart blood yield the bacillus. There is extensive subcutaneous gelatinous œdema notable in its absence of blood staining, and gas formation. There are no microscopic lesions in the organs, and the bacillus does not form filaments on the peritoneum. B. œdematiens is also pathogenic for rabbits.

Immunity.—The Medical Research Committee[31] record that an antiserum can be prepared to the toxin of B. œdematiens capable of affording protection against it. In three months an antiserum containing 5,000 units per c.c. was obtained.

BACILLUS HISTOLYTICUS (Weinberg and Séguin, 1915.)

This bacillus was isolated from war wounds in man. In nature it has been found in garden earth. It derives its name from its power to liquefy the living tissues of infected animals.

Morphology.—It is a gram-positive, motile rod, measuring 2·5 by 0·7 microns, shewing oval, subterminal or central spores, and with a tendency to involution forms in cultures over twenty-four hours old.

Cultural Characters.—On the surface of blood agar dewdrop-like colonies are formed which later become flat with crenated edges. In deep agar the colonies are compact and lobulate or fluffy, In meat media it causes opacity and gas formation. and the meat becomes digested. Balls of tyrosein are produced. Gelatin is liquefied. It ferments glucose, lævulose, and maltose very slowly, and the production of acid is weak. It is actively proteolytic. The gas in meat medium comes from the protein, and is formed in the absence of carbohydrate. B. histolyticus is therefore an example of a putrefactive organism which is sometimes pathogenic. In this respect it lies between the pathogenic non-proteolytic anærobes which have been dealt with and the non-pathogenic proteolytic B. sporogenes still to be mentioned. It produces a filterable toxin in the early stages of growth in culture. In identifying this organism its property of tissue liquefaction rather than its morphology and cultural characters should be taken into account.

Pathogenicity.—The action of this anærobe upon living tissue is unlike that of any other organism. In inoculated guinea-pigs the skin and muscles are dissolved away, leaving nothing but the bones and tendons; or the wall of the abdomen may be gone, leaving the intestines protruding. The organism may or may not be recovered from the heart blood. Beckwith and MacKillop[36] record that histological examination of the liver, spleen, kidney, lung, suprarenal, and heart muscle reveals profound degenerative changes. There seems to be only one record of the occurrence of this organism in animals. Weinberg and Séguin[35] found it in the above-mentioned horse along with B. œdematiens.

Many other anærobic sporulating bacilli are to be found in nature, and have been isolated from wounds in man, but those mentioned are the only known ones of interest to the veterinarian as being pathogenic for animals or man from their ability to multiply in the living body. I now wish to refer briefly to the best example of the non-pathogenic putrefactive bacteria by way of contrast.

B. Sporogenes (Metchnikoff, 1908.)

This organism is of interest to veterinarians in that to this group belongs the "Reading bacillus," which has been used to some extent in veterinary practice in the treatment of sloughing wounds.

The B. sporogenes is a large, gram-positive, actively motile bacillus of varying length, occurring singly, in pairs, or in chains. Morphologically, it is the same as Vibrion septique. It presents marked features which serve to distinguish it in culture from the pathogenic anærobes. In meat medium there is a strong growth and the meat is digested and blackened with the production of gas with a putrid odour. Whereas the pathogenic anærobes show no inclination to attack proteins, the B. sporogenes shows this as a main feature.

B. sporogenes is one of the commonest anærobes in nature, and is to be found in the alimentary canal of all species. It is the commonest contaminator of stock laboratory cultures of pathogenic anærobes, and is responsible for the many incorrect descriptions of Vibrion septique and other pathogenic anærobes as putrefactive organisms.

Bacillus Botulinus (Van Ermengem, 1896).

I now wish to review the question of botulism from the point of view of the veterinarian. The question of whether botulism occurs naturally in animals as it is known to occur in man is one of considerable interest, both to veterinarians and medical men. In this paper I do not intend to deal with so-called "grass disease" in horses which has been thought by some to represent botulism in that species, since an investigation of "grass disease" is in progress, and the experimental work has not matured as yet into results which it would be advisable to publish. I wish, however, to examine the evidence for and against certain disease symptoms in animals being botulism.

Morphology.—This bacillus is a gram-positive, sluggishly motile, sporulating pleomorphic rod, occurring singly or in pairs or chains. It closely resembles the Vibrion septique and B. sporogenes under the microscope. The spores are oval, central or subterminal, and are formed more readily by some strains than by others. In view of the likeness in general microscopic appearance of this bacillus to Vibrion septique and B. sporogenes the morphology of the organism cannot be accepted as a criterion for identity.

Cultural Characters.—B. botulinus is a strict anærobe. On blood agar minute round grey colonies develop shewing hæmolysis. In deep agar the colonies are lens-shaped or kidney-shaped. In meat medium the liquid becomes turbid and the meat blackened and digested. Gas is formed and there is an odour of putrefaction, but not so disagreeable as from B. sporogenes, whose action on the meat is somewhat quicker and more complete. Milk is firmly clotted without gas production. This organism is a formidable producer of toxin in *pure* culture. Certain anærobes in mixed culture inhibit toxin production (Hall[29]). The view of the Medical Research Committee[31] that this organism shows predominant saccharolytic properties as compared with its proteolytic properties is held by later workers to be wrong. Hall[29], Kahn[37], and Wagner, Dozier and Meyer[38] find it predominantly proteolytic, or in other words, an organism belonging to the putrefactive group, although producing a powerful toxin. It ferments glucose, maltose, and glycerin. It resembles B. sporogenes in not fermenting lactose and saccharose. Growth occurs at summer temperature as well as at blood heat. Reddish examined nineteen strains of B. botulinus as received from American laboratories, and found eighteen of them contaminated with B. sporogenes. He believes the impurity of American strains to be due to the fact that they were mostly obtained from spoiled foods, whereas European strains were not; for example, Ermengem's hams did not smell and Madsen's fish had only an intense butyric odour.

Distribution in Nature.—The bacillus botulinus has been shewn by Meyer and Dubovsky[48] to be a common soil anærobe in America, and they found the Type B bacillus more common in cultivated soils than Type A. Botulinus spores were more common in virgin soils than in manure from cattle and pig pens. They have shewn the presence of these spores in the soil of Canada, England, Belgium, Denmark, Netherlands, Switzerland, and other countries, and have found them on the pods of string beans, leaves, mouldy hay, ensilage, and decayed vegetation. They conclude that from a practical standpoint the B. botulinus is ubiquitous. In the A.D.R.A. laboratory several soil samples from Northumberland have been examined, and the presence of B. botulinus spores demonstrated. Tanner and Dack[42] conclude that the B. botulinus is probably a common saprophyte widespread in nature. It has been shewn that B. botulinus is not infrequently a common inhabitant of the intestinal tract of healthy domestic animals.

Pathogenicity.—The question naturally arises whether the B. botulinus and its toxin are, separately or combined, the cause of disease symptoms in animals. Experiments show that the bacillus minus its toxins when injected or fed to animals, except in overwhelming numbers, is harmless. The toxin of the B. botulinus is very powerfully toxic. Madsen's filtered toxin was fatal to guinea-pigs in a dose of ·0015 c.c. The botulinus toxin is different from that of other organisms dealt with here, B. welchii, Vibrion septique, etc., in that the botulinus toxin increases in quantity with prolonged incubation, whereas that of the other bacilli is an early product of multiplication and decreases in quantity if incubation is prolonged after eighteen to twenty-four hours. The toxin of B. botulinus is relatively stable. The toxin obtained from B. welchii and the others produces an instant effect upon intravenous injection into rabbits, whereas that of B. botulinus only acts upon the system after a period of incubation of some hours. The botulinus toxin is the only known toxin which is pathogenic *through the mouth*, indeed it is only upon the production of such a toxin that one places a bacillus in the botulinus group. The B. botulinus has been divided into types according to the toxin it produces. Types A, B, and C have been recorded. The antiserum for any one type does not protect an animal against the toxin of any other type. Seddon's parabotulinus bacillus[39] is probably identical with Type C. The toxins of all the types are pathogenic upon ingestion. Since the spores of B. botulinus have been shewn to be ubiquitous they must be taken into the alimentary canal of man and animals on numerous occasions. Coleman and Meyer[40] found that *enormous* numbers of toxin-free spores were required to infect guinea-pigs when fed. Over 2,000 billion spores might be negative in result, and at other times eleven billions infected, and they conclude that "unless introduced in enormous numbers we have little to fear from the ingestion of toxin-free spores."

It is known that when the B. botulinus finds suitable anærobic conditions for nutrition, it will multiply outside the body and produce its deadly toxins. It cannot produce its toxins outside the body unless it can find such conditions. In the case of the human being these suitable conditions are found in tinned and bottled foods when the spores of the bacillus have escaped death in the sterilisation processes of the foods' manufacture. In chickens, Hart[14] has recorded botulinus poisoning where jars of spoiled string beans were fed to them. In horses so-called "forage poisoning" in America has been attributed by some observers to botulism, from toxin produced in spoiled fodder.

While it can readily be understood that botulism in man and fowls, and possibly in animals, arises in the afore-mentioned manner from opportunity being given the bacillus to multiply outside the body, the question of the bacillus multiplying in the living tissues and fluids of man or animals and producing botulism is more than doubtful.

P. F. Orr[41], who found that washed (toxin-free) spores are able to produce botulism by feeding or injection and that the bacillus can be recovered from internal organs of animals dead after feeding or injection, concludes that the B. botulinus will grow and produce toxin in the guinea-pig. It is curious, however, that Orr further concludes that botulism due to the ingestion of spores is probably very rare, if it occurs at all.

Tanner and Dack[42] found no difficulty in finding the organism in the brain of animals shewing latent death, and they confirmed the organism as B. botulinus by growing it in brain medium and finding the culture pathogenic for guinea-pigs by feeding. Bachmann and Haynes[43] isolated B. botulinus from the liver of a guinea-pig killed by feeding B. botulinus. Coleman and Meyer[40] conclude that "toxin-free spores germinate" in the body, "and the vegetative forms arising from this germination multiply and liberate toxin in the animal body." Hall[29] made heart blood cultures from all of his experimental animals dying of botulism, and spleen and liver cultures from many of them, without being able to cultivate the organism. Hall and Davis[44] find themselves unable to agree with Coleman and Meyer's conclusion, which they think is based upon insufficient evidence. In Hall and Davis' Table III., shewing eighteen guinea-pig inoculations, the B. botulinus was isolated from the heart blood of two guinea-pigs. Commenting on this they say "this is not a common occurrence, but has been recorded in the literature. The animals had been dead some hours before examination, and there is no satisfactory evidence that the organisms were circulating in the blood at the time of death." Hall and Davis inoculated two guinea-pigs with culture, and immediately following death aspirated several cubic centimetres of blood from the heart, but both samples were negative in culture.

Graham and Schwarze[45] record that "evidence of growth *in vivo* with elaboration of toxin has not, in our studies, been observed."

Tocher[46] records that he has been able to cultivate an organism "morphologically identical" with the B. botulinus from the spleen of every case of so-called "grass disease" in equines in Britain. The words "morphologically identical," however, cannot be accepted as showing his organism to be the B. botulinus, since it is well-known that this organism is indistinguishable morphologically from the Vibrion septique, B. histolyticus and B. sporogenes. Tocher's organism was, apparently, innocuous when *fed* to animals, and for this reason alone cannot be accepted as B. botulinus. That the organism shall produce a toxin pathogenic by ingestion is the *sine qua non* in establishing its identity as B. botulinus. Hall[29], on this point says, "the animal (feeding) test constitutes so far the only single certain method of recognition of B. botulinus in view of the morphology and cultural similarity of B. sporogenes and B. histolyticus. No other bacterial toxin is fatal orally, and no other anærobe is known that is pathogenic when fed," thus putting into words what is now generally accepted by the scientific world regarding the B. botulinus. Tocher's recorded method of examining the spleens of "grass disease" horses for the B. botulinus was to dip the spleen into boiling water for three minutes, with a view to sterilising the splenic capsule preparatory to cutting through it in order to obtain pulp to sow cultures with. The spleen of "grass disease" horses is frequently four times the size of a normal horse's spleen, and it would appear as though the temperature of water into which such an organ was dipped must drop many degrees below sterilising temperature even for non-sporulating organisms. This technique doubtless accounted for the growths obtained of what may have been the Vibrion septique and B. sporogenes in Tocher's culture media. The illustrated lesions in a guinea-pig dead from inoculation with Tocher's organism were not lesions such as the B. botulinus causes in animals dead from inoculation or feeding, but were a hæmorrhagic subcutaneous œdema such as is caused by an organism of the Vibrion septique type.

I have recently carried out some experiments to see (1) if the B. botulinus multiplies in the bodies of animals inoculated with whole culture; (2) if the horse is a relatively susceptible animal to infection or intoxication from B. botulinus culture, and (3) if the symptoms and post-mortem appearances resemble those found in "grass disease" horses. This series of experiments is still in progress, and as part of the "grass disease" investigation will fall to be dealt with fully in an article upon that subject at some future time, some of the experiments may be usefully recorded here as bearing upon what is under discussion.

Shetland Pony, No. 262.—Was fed by stomach tube at 11 a.m. on 26th June, 1923, with 6 c.c. forty-eight hours whole culture of B. botulinus, Loch Maree strain. The same culture was fed at 10-30 a.m. on the same date, dose 1 c.c., to *Control Guinea-pig* 270. The guinea-pig died at 10 p.m. on the 26th June, but the pony did not show any sign of illness, and was disposed of several weeks later in perfect health.

Clydesdale Farm Horse, No. 274.—Was inoculated subcutaneously on the side of the neck at 4 p.m. on 26th June, 1923, with 3·5 c.c., forty-eight hours whole culture of B. botulinus, Loch Maree strain, from the same culture flask as Pony 262, and *Control Guinea-pig* 270. On June 27th, 28th, and 29th, this horse was in perfect health, but on 30th June it did not seem to be perfectly well, although nothing very definite could be detected. This impression remained regarding the horse, which was turned out to grass, until July 3rd, when the animal was found paralysed in the field with the tongue hanging out and salivating. The symptoms were those of human botulism, and no clinical resemblance could be detected to "grass disease."

Pony No. 333.—Was fed by stomach tube at 11 a.m. on 17th March, 1924, with 23 c.c. of an eleven days' whole culture of Madsen's B. botulinus. The pony remained in perfect health. The same pony was fed by stomach tube at 11 a.m. on April 1st, 1924, with 250 c.c. of a seven days' whole culture of Madsen's B. botulinus. It was found paralysed at 8 a.m. on April 3rd, 1924, and died at 9 a.m. The tongue was not hanging out, and salivation was not present. Post-mortem examination showed no lesion except a slight blush of congestion on the gastric mucosa. Cultures upon anærobic media, from heart blood, spleen, and liver were all negative. *Control Guinea-pig No.* 340 received by the mouth 3 c.c. of the seven day culture from the same flask as the 250 c.c. dose for pony 333, and was found dead within twenty hours. Cultures from the heart blood and liver upon anærobic media were negative. The main lesion was hæmorrhage into the pleural cavity.

Guinea-pig 343.—Received intraperitoneally 1·25 c.c. from the same culture flask as *Guinea-pig* 340, and was found dead within twenty hours. The main post-mortem lesion was hæmorrhage into the pleural cavity. There were also a few small hæmorrhages into the stomach wall. Only one or two single bacilli were found by the microscope in a liver impression preparation. Cultures from the heart blood and liver substance were negative.

Pony 357.—Was inoculated subcutaneously on the side of the neck, on April 16th, 1924, with 2 c.c. eighteen days whole culture of Madsen's B. botulinus. A slight local œdema was present in twenty-four hours, 3 inches in diameter, and ·5 inch thick, and this disappeared in a further twenty-four hours. The pony exhibited no symptoms of illness, indeed from the condition of skin and bone in which it was purchased on April 14th, it developed splendid condition. On May 6th this pony was given subcutaneously 10 c.c. of the same culture, but passed through a filter candle to free it of bacilli and spores.

No symptom of illness resulted. As a control to Pony 357, *Guinea-pig* 358 was used.

Guinea-pig 358.—Was fed on April 16th, 1924, 2 c.c. of whole culture from the same flask as used for Pony 357, and death occurred in nineteen hours. Cultures made from the heart blood and liver upon anærobic media were negative.

Pony 386.—Was inoculated subcutaneously on the side of the neck at 3 p.m. on May 20th, 1924, with 20 c.c. eighteen days' whole culture of Madsen's B. botulinus which had been allowed to settle, but nothing more than a local œdema resulted.

These experiments in so far as they have gone, at least indicate (1) that the B. botulinus shows no invasive powers in the animal system; (2) that the horse is comparatively insusceptible to botulism, both when culture is fed and inoculated; and (3) that the symptoms and post-mortem appearances in horses which become intoxicated and die as a result of feeding or inoculation with culture show nothing comparable with "grass disease." They would further lend support to the view that the B. botulinus does not multiply in the tissues or fluids of the living animal, nor produce its toxin there. This brings the B. botulinus still nearer to the putrefactive B. sporogenes group, and removes it further from the tissue-invasive Vibrion septique, B. welchii, and B. chauvœi. There remains to be considered whether the B. botulinus finds suitable conditions of nutrition, anærobiosis, etc., in the alimentary canal of animals to enable it to multiply and produce its toxins there, the toxins then becoming absorbed and causing symptoms and death. I have shewn how it has been established that the B. botulinus is practically ubiquitous in its distribution, and how it has been shewn to be a common inhabitant of the alimentary canal of healthy animals. One would appear to be justified in concluding, therefore, that the bacillus does not, as a general rule, multiply and produce toxin in harmful quantities in the alimentary canal, or there would be no human or animal population left on the earth. There is the possibility, however, that the bacillus under certain circumstances may find its environment in the digestive tract suitable for growth and toxin production, to an extent sufficient to cause symptoms in the host. Mitchell[47] in a Preliminary Report on Equine Botulism in Canada records that he took material from the stomach and bowel contents, and from it grew anærobes in artificial culture which killed guinea-pigs and horses by feeding. Chickens were immune to feeding with these cultures, and from this Mitchell concluded he was dealing with the type B. bacillus. In order to prove that the spores of botulinus had passed through the chickens Mitchell replaced the chickens by guinea-pigs which were fed from the fæces-soiled floor of the chickens' cages. The guinea-pigs died as a result, and this was accepted as proof of the presence of spores in the chickens' excrement. Mitchell's work does not show the disease in horses to have been equine botulism. It merely shows that the spores of B. botulinus were present in the stomach and bowels of the horses as they are present in healthy horses, and further that these spores will germinate and produce toxin in culture outside the body. The fact that guinea-pigs died when fed off a fæces-covered floor is not proof of botulism; it merely emphasises what everyone who keeps guinea-pigs knows to his cost—that they die, if they are not kept in scrupulously clean cages, from a kind of paratyphoid. If one is to show that a disease of animals is botulism one must show that the alimentary canal of animals as soon as they are dead or animals killed when suffering from symptoms, contains the toxin of the B. botulinus. In this connection, Dubovsky and Meyer[48] have demonstrated the presence of toxin in the crops and intestines of limberneck fowls, and give their opinion that "toxin tests alone support the clinical diagnosis of botulism," on account of the wide distribution of this organism in the soil. They further give their view that "an etiologic relationship of these organisms to the diseases or lesions from which they have been isolated is not proved," and that "in future studies an attempt should be made to demonstrate the toxin in the suspected feed or in the gastric contents of the animals which exhibit symptoms of botulism," views with which I am in full agreement for the reasons given. Graham and Boughton[49] found the specific toxin of the Type C bacillus in one chicken out of five outbreaks of disease which they attribute to botulism Type C. In four of the five outbreaks the numbers of deaths were 8, 60, 27, and 15. In these outbreaks the suspected feed was not definitely incriminated. These appear to be the only instances upon record of finding botulinus toxin in the alimentary canal of animals, or birds suspected to have died of botulism. There are other instances of supposed botulism where the diagnosis cannot be accepted as conclusive. Graham[50] writes an indefinite article upon "Botulism in Swine and its relation to immunisation against Hog Cholera," but largely withdraws his remarks in a subsequent paper. Graham and Schwarze[45] record: "B botulinus Type A, associated with a fatal pasture disease of horses," and place on record that "several specimens, including spleen, brain, and intestinal contents, together with a sample of hay from the pasture in which the horses had died, were subjected to bacteriologic study. One spleen proved to be contaminated with an anærobe indistinguishable from B. botulinus A. They record that the significance of this finding is not definitely concluded. Graham and Boughton[49] record two outbreaks of disease in chickens likened to "limberneck," in which the Type A bacillus was encountered. One of these was "presumably" from feeding garbage, and in the other, poisonous feed could not be established. They found the Type B bacillus in one of three such chickens, but could establish no relationship between the bacilli and the disease, since even artificial cultures induced no illness. They also found the Type C bacillus in the intestines "or internal organs" of one or more sick birds in five such outbreaks. In order therefore to establish an outbreak of disease as botulism

it would seem to be necessary to establish that the feed is toxic upon ingestion or show the presence of toxin in the alimentary contents of recently dead animals. Dickson[51] records an outbreak of botulism in man, and how chickens which ate the remnants of the food responsible became paralysed and died. Hart[4] investigated an outbreak of disease in chickens where 643 died in four days. The cause was believed to be due to spoiled string beans, of which four jars were thrown in the garbage pail. Diagnosis of botulism was made from the clinical symptoms. This is an outbreak which, from the history of feeding spoiled beans, can readily be accepted as probably botulism, but no experimental work seems to have been done to prove it. In the many cases of human botulism there is some history of spoiled food. For example, three people died who had partaken of a particular cheese in America. Nevin[52] found the toxin of the B. botulinus in portions of the uneaten cheese. In the recent outbreak in human beings at Loch Maree, in Scotland, only those were affected who had partaken of wild duck paste from a particular container, and the toxin of B. botulinus was shown to be present in the uneaten remains of the paste. In South Africa, Theiler[53] has shown lamziekte to be due to a bacterial toxin present in bones which the cattle licked. He found this toxin in the carcases to be toxic by ingestion, and as such the organism which produced it would come under the botulinus group. Theiler has not yet recorded that he has isolated and studied this toxigenic anærobe in pure culture. In this connection we have to note that Seddon[39] found a toxigenic bacillus in the bones of a cow which had died of Midland cattle disease in Tasmania, and lain skinned and exposed in the open for four months. Seddon does not attempt to show that the bone marrow contains a toxin which is toxic by ingestion, but he does show that the marrow contains a toxigenic bacillus whose toxin from cultures is fatal upon ingestion. Possibly Theiler's toxin in carcases is formed by the toxigenic bacillus which Seddon has shewn can be present. In this connection it is interesting to note that Graham and Boughton[49] have found that an antitoxin from three of their chicken strains of Type C, B. botulinus, protects guinea-pigs against lethal doses of Seddon's B. parabotulinus. Graham and Boughton have found Type C in the intestines, " or internal organs " of sick chickens, and shewn it to be the Type C of Bengston who first isolated this anærobe from the larvæ of Lucilia cæsar, and found that it produced a soluble toxin fatal upon ingestion though with cultural differences from Types A and B, and that its toxin is not neutralised by the antitoxins of Types A or B. Seddon's B. parabotulinus would, therefore, appear to be identical with Bengston's Type C from the larvæ of L. cæsar.

Theiler in his work upon lamziekte shows that blow-fly larvæ from lamziekte carcases are toxic to cattle upon ingestion, and that mixed toxic cultures can be obtained both from the larvæ and pupæ, though he has also shewn that the toxin is generated in the spleen of an unopened carcase after putrefaction has been going on for twenty-four hours, the organism presumably penetrating to the spleen from the intestinal contents after death.

Possibly the anærobe found in L. cæsar larvæ in America, the anærobe which Seddon found in exposed bones from Tasmania, and the anærobe producing toxin in carcases in lamziekte areas in South Africa are all one and the same Type C, B. botulinus.

Seddon accepts his bacillus as the cause of Midland cattle disease. He does not show that the bones contained the toxin and that the disease is caused by ingestion of this toxin from the carcases, but would seem to suggest that the bacillus he found in the bones four months after death was there during life, causing the disease. This hardly seems to be justified by his observations. He says, however, that " other animals had previously died of the disease in this paddock."

Limberneck in poultry seems to have received some acceptance in America as being botulism. An excellent article by Wilkins and Dutcher[54], however, is noteworthy in this connection. They studied limberneck in chickens alongside experimental botulism in chickens, and conclude that the two conditions are markedly distinct. They show that the temperature of limberneck fowls is raised, whereas that of botulism fowls is sub-normal. They find, however, that true limberneck symptoms can be produced by feeding to chickens the larvæ of L. cæsar developed from eggs laid upon limberneck carcases. This does not necessarily incriminate the B. botulinus.

The general conclusion one is forced to regarding botulism in animals or poultry is that the B. botulinus is a saprophyte rather than a parasite, that it can only be accepted as the cause of symptoms where the history of an outbreak shows that its toxins have been formed outside the body in spoiled food or carcases, that there is insufficient evidence as yet to inculpate the organism as the causal factor in diseases of animals by growing and producing its toxins in the alimentary canal, and that the weight of evidence is against the B. botulinus being able to grow and produce its toxins in the tissues and fluids of the living animal as true pathogenic organisms do.

I trust my paper will stimulate an interest in such anærobic infections on the part of the general practitioner and lead him to make greater and greater use of the assistance which the laboratory observer can undoubtedly give him.

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Fulham Veterinary Surgeon Libelled.

Teacher's Strange Letter.

Kathleen Everett, 30, school teacher, of Napier Avenue, Hurlingham, pleaded not guilty before the Common Serjeant (Sir Henry Dickens, K.C.), at the Old Bailey, on November 11th, to a charge of publishing libel on Arthur Pinson Case, veterinary surgeon to the Fulham Borough Council. She also pleaded justification, and handed in a plea which the judge, after reading, ordered to be struck out.

Mr. Travers Humphreys said that the only question for the jury was whether the document was a libel. The prosecutor was a M.R.C.V.S., and a veterinary surgeon to the Fulham Borough Council. When he was away from home through illness a dog belonging to the defendant was treated at his surgery. Defendant then wrote the letter, which was the subject of this charge, to one of the members of the Fulham Borough Council. It alleged that prosecutor was in disgrace at Red Lion Square, which was the head-quarters of the R.C.V.S., and also with the R.S.P.C.A. "If an animal needed attention during the night," the letter continued, "he would rather kill it than help it. If the poor dumb creatures could only speak of their terrible sufferings from such villains as Case, laws would long ago have been made to save them from unqualified men and scoundrels."

Mr. Case gave evidence that he was away from home through illness when the defendant's dog was treated by his assistant. He knew nothing about the defendant until she came to his office and abused him.

Defendant then went into the witness box and gave a detailed explanation of her reasons for writing the letter.

After she had been speaking for some time, a juryman asked the judge if they were to go on listening to her.

The Judge: If you can stop this lady, you can. I can't.

Defendant was found guilty, and Mr. Travers Humphreys then said that the prosecution was brought by Mr. Case to put a stop to "this impossible state of things." Defendant had made speeches about him at street corners. She had also written a letter to the detective-sergeant in charge of the case threatening to shoot him and other people "who were helping to blacken her father's name."

The Common Serjeant postponed sentence until next sessions, and ordered the defendant to be kept under observation.—*West London Observer.*

Marriage.

Edwards—M'Fadyean.—On the 7th November, at Emmanuel Church, Bombay, by the Rev. L. Butcher, James Thomas Edwards, Indian Veterinary Service, to Constance Amy, daughter of Sir John and Lady M'Fadyean, of the Royal Veterinary College, London.

The next Congress of the Royal Sanitary Institute and Health Exhibition will be held in Edinburgh from July 20th to 25th, 1925, by invitation of the Magistrates and City Council.

The Veterinary Record.

29th November, 1924.

JAUNDICE IN DOGS.

Amongst the many unsolved pathological problems in the dog which await solution, that of jaundice is by no means one of the least interesting or important. The annual loss in numbers from this disease alone must be very great indeed, and probably runs into many thousands, for it must be the common experience of every practitioner that even amongst the cases which are brought for treatment more die than recover. Many thousands of cases are never seen by a veterinary surgeon, and of these very few indeed regain health and strength. In hounds alone the losses can be valued in tens of thousands of pounds. It does not seem to redound to the credit of our profession that we have not yet been able to recommend any form of successful treatment or even to suggest any measures which have proved effective in preventng the disease. Our lines of treatment have been empirical and we have taken widely divergent views as to the probable cause or causes, while in the majority of cases the disease has been so rapidly fatal as to admit of little careful observation and experiment. The layman, and especially the kennelman, has always looked upon jaundice as a symptom of, or sequel to distemper, and the same belief is still held by many veterinary surgeons; but though jaundice may follow distemper (and it very often does) the two diseases are separate and distinct, and should not be confused the one with the other. This view has been gradually accepted during the last twenty-five years by careful observers, and even to-day there are omniscient "quacks" and other "canine specialists" who subscribe to it. These latter are ready enough to promise a certain cure in the case of distemper, but wisely refrain from advertising their ability to cure jaundice, knowing full well that it is an entirely different problem. The late Wallis Hoare held the opinion very strongly that this disease—he named it enzootic jaundice—was not one of the manifestations of distemper, but rather that it was related in its cause to such diseases as red-water and piroplasmosis, and he was strengthened in his opinion by the discovery of the piroplasm in East African malignant jaundice in dogs. His experiments with trypanblue, however, were unsuccessful and caused him great disappointment. Nevertheless, he wrote confidently in 1912, "Further research will probably prove that this scourge depends upon some parasite in the blood." His prophecy seems likely of fulfilment, for on another page of this issue there appears a preliminary note by Messrs. C. C. Okell and T. Dalling on the results of research into the disease which give promise, at the very least, of some measure of success in the elucidation of this difficult problem. These gentlemen will undoubtedly receive the thanks and encouragement of every general practitioner and every dog owner in the work that they are doing. But even when the causal organism is found, there is much remaining to be explained. There is the definition of the intermediary host—possibly the tick, louse, acarus, flea, or fly. There is yet to be explained why the incidence of the disease is greater in the country than in the town, a question which may be related with that of the intermediary host. Information is wanted as to why there should be such great differences in the appearance of the organs in different *post-mortem* examinations, and why invagination of the bowel should so frequently be found after death, when such a condition could scarcely be diagnosed with certainty during life and is not commonly found except in cases of jaundice.

Great as is the need for research into distemper, the need for research into jaundice is almost as necessary, for huntsmen and kennelmen know only too well that large as the loss from distemper may be, the loss from jaundice is even more certain and is impossible, in our present state of knowledge, to prevent.

World Dairy Congress for England.

At a recent meeting of the Central Council of Milk Recording Societies, acting in conjunction with the Ministry of Agriculture, it was stated that a request of the Ministry that a representative of the council should be appointed to attend the World's Dairy Conference, to be held in this country in 1926 or 1927, had been considered; and the proposal of the council was that Mr. S. Wallace should be their representative.

Mr. Wallace stated that he had attended a preliminary meeting of the committee at the Ministry, and all interests were represented. It was estimated that the cost would be about £10,000, and the Government would assist in providing the funds. He moved that they give this movement every possible support.

Mr. E. W. Langford thought the conference would cost at least £20,000. The last congress was held in America, and it was attended by over 1,000 delegates from all over the world, and cost £27,000. A congress here on the same lines would arouse tremendous interest in America. At present 67 per cent. of the world's exportable milk produce was sent to this country; and we only produced 46 per cent. of our supply. He seconded the motion, and it was agreed to.

CLINICAL REPORT.

Sub-Acute Obstruction or Impaction of the Cæcum in the Horse.

By H. Caulton Reeks, F.R.C.V.S., Spalding.

In English veterinary literature there are not many recorded cases of impaction of the cæcum of the horse. In fact, since it was first clearly described by Gofton in 1912, and afterwards mentioned in my book *Common Colics of the Horse* in 1914, I do not remember having seen another case recorded. It must, therefore, one would think, be most rare; that, in part, is my reason for reporting this case.

Not solely on account of their rarity, however, are these cases interesting. When, after a period of years spent in treating the more or less common forms of obstruction, the practitioner finally encounters a case of cæcal impaction, he will find that such cases have several extremely interesting characteristics of their own.

They are interesting, in the first case, on account of the long time the patient lingers on with little apparent constitutional disturbance. In the case I am recording, the animal lived for so long as seventeen days before being shot. Secondly, the condition is interesting on account of the ease and comparative certainty with which it may be determined during the life of the patient. One is afforded an opportunity of making this certain diagnosis by reason of the condition always running the protracted course to which I have referred, for, during this course, the colon slowly becomes more or less empty and manual exploration comparatively easy. Diagnosis is then rendered certain by each succeeding exploration revealing the hard and impacted cæcum, recognisable by its shape, by its being found regularly and consistently in the same position, and showing no alteration in the consistency of its contents.

They will prove further interesting, I am afraid, if I may judge from this case in particular, in that they will show the utter futility of all ordinary therapeutic measures, including constant lavage of the system by means of the stomach tube and by enemas. Sufficient, perhaps, is not yet known of this condition to allow one to state flatly that recovery may never occur; but it seems plain that, at the most, recovery can only be occasional, and that the condition once diagnosed may be regarded as fatal, the case slowly running on to a combined enteritis and peritonitis, with bacterial intoxication of the blood stream, weakening of the intestinal walls and, finally, rupture. In fact, rupture of the cæcum itself was found in the autopsy in each of the four cases reported by Gofton, and rupture of the colon in a similar case recorded by myself. In connection with this point, the case I am reporting was again interesting in that there was no sign of rupture. True, the patient was shot, but she was plainly in the last stages of dissolution when this was done.

I will now give the history of the case and the treatment the patient received, but will endeavour to be brief.

Friday, October 10th.—The subject, an aged Shire mare, bought by the owner only three days before she was taken ill, was seen on this date by Mr. H. C. Reeks, jun. There was a history of dull pains the whole of the previous day. At no time were they alarming, but the mare for the whole time took neither food nor drink. Impaction of the colon was diagnosed, but rectal examination failed to confirm this. As it was evident, however, from the symptoms that impaction existed, the usual treatment was adopted, namely, the administration of eight drams of aloes, balls of ammonium carbonate and nux vomica, and capsules of turpentine and methylated ether. This was in the morning.

In the afternoon the same treatment, with the exception of the aloes, was repeated, and the administrations of enemas commenced. A drench containing ether, turpentine, and linseed oil was left to be given later in the evening.

Saturday, the 11th.—Morning.—I saw the case myself and was informed that no action of the bowels had taken place. The mare was still in dull pain. No rectal examination was made. I assumed the case with which we were dealing was one of those we sometimes meet with, namely, intestinal impaction, in which the aloes had failed to act. I administered hypodermically two grains of arecoline hydrobromide.

Afternoon.—Mr. Reeks, jun., and I saw the case together. Asking whether or not there had been any response to the arecoline we received a very emphatic and delighted reply in the affirmative. The interpolated adjectives prevent its exact quotation. Robbed of these, it amounted to the fact that there must have been at least thirty defæcations commencing shortly after I had left in the morning and continuing for two or three hours thereafter. The mare took the water we offered her and was picking interestedly at her food. We both thought we had seen the end of the case and crossed it from our visiting list.

Sunday, 12th.—The mare was easy all day and picking occasionally at her food. In the evening the owner again requested our attendance. Mr. Reeks, jun., visited again, administered the usual stimulants, and advised enemas during the night. On his return he explained to me the continuous nature of the dull pains which were still in evidence, together with a significant character of the pulse which, though in no way troubled, was nevertheless greatly hurried. This character of pulse, I may add, was maintained throughout the case. For several days, in fact, any casual observer might have walked into the mare's box, seen her picking about in the manger, and very naturally have concluded there was nothing seriously wrong. He would, however, have been misled by the mare's quiet attitude; for at any one time, whenever the pulse was taken, it was found to be hammering away at the rate of 100 per minute.

At this stage it was easy for both of us to say (*a*) the small colon and the large are now practically empty; (*b*) judging from the mare's symptoms it is still quite evident there is impaction; (*c*) the whole thing points to the cæcum being the seat of trouble.

Monday, 13*th*—From now on there is no need to detail the symptoms. What was in existence on the 12th was practically a description of the case as it was seen on each succeeding day, namely, inappetence, eating but very, very little, refusing even water except as she was seen playing with it with her lips, and the continuously hurried pulse with practically no exhibition of pain. There were occasional actions of the bowels, plainly nothing approaching the normal, but just so much as one would expect considering the small amount of food the mare was taking. Mr. Reeks, jun., all along kept control of the case, while I was in occasionally, more or less as an interested observer. To-day the use of the stomach tube by way of the nostril was commenced, and the mare received:—

Morning.—Sodium chloride, 1lb. in three gallons of warm water. *Evening.*—Ditto.

Tuesday, 14*th*—*Morning.*—Ditto. *Evening.*—Ditto. It was on the evening of this day that Mr. Reeks, jun., returned saying he was convinced the case was one of cæcal impaction, and that on rectal examination the impacted organ could now be felt, this by reason of the empty state of the other bowels.

Wednesday, 15*th*—*Morning.*—Sodium chloride in three gallons of water. *Evening.*—Ditto; also balls of ammonium carbonate and nux vomica.

Thursday, 16*th*—*Morning.*—Sodium chloride in three gallons of water. Balls of ammonium carbonate and nux vomica. *Evening.*—Sodium chloride in three gallons of water.

Friday, 17*th*—*Morning.*—Sodium chloride in three gallons of water. *Evening.*—Sodium chloride in three gallons of water. Balls of ammonium carbonate and nux vomica.

Saturday, 18*th*—*Morning.*—Sodium chloride in three gallons of water. *Evening.*—Sodium chloride in three gallons of water.

Sunday, 19*th*—Until now we had been hoping that, with the continued flooding of the intestinal tract with water, the contents of the cæcum, if only from absorption and not from the water reaching it mechanically, would eventually soften and allow the bowel wall to contract; for not only had the stomach tube been regularly used for seven days, but in addition the owner all this time was repeatedly giving enemas.

No change, however, had occurred and although treatment was continued as a forlorn hope, it was more or less realised by all of us that the case could only have a fatal termination. The stomach tube was again used, but to-day the quantity of fluid administered was more than doubled, approximately eight gallons of water being injected at each administration, as under:—

Morning.—Sodium chloride, 1lb. in eight gallons of water. *Evening.*—Ditto.

Monday, 20*th*—*Morning.*—Sodium Chloride in eight gallons of water. *Evening.*—Sodium chloride in eight gallons of water.

Tuesday, 21*st*—To-day only one visit was paid, and the usual quantity of sodium chloride and water injected.

Wednesday, 22*nd*—Again only one visit was paid. This was in the afternoon, and again the same quantity of saline solution administered. On this occasion I made a rectal examination myself and was quite easily able to feel the impacted cæcum. Here I would like to explain that, so far as my own experience teaches me, there is only one other condition with which it might be possible to confuse it, and that is a calculus in the large colon of similar size and shape.

I know now, however, that these two conditions may be differentiated in the following manner. The impacted cæcum is to be recognised more or less by its shape, by it being not quite so resistant to the touch as a calculus, but more by reason of the fact that always at each examination it is found occupying the same spot, namely, well at the full length of one's arm, and well to the right and centre. In other words, it is a fixed object which, if felt at all (during periods of straining it sometimes cannot be reached) is always in the same position and cannot be moved. A calculus, on the other hand, is inclined to change its position, and at such times as it is nearer the rectum can with the exploring hand be pushed in front of one.

Thursday, 23*rd*—*Morning.*—Eight gallons of saline solution injected. *Evening.*—Eight gallons of saline solution injected. In addition 1½ grains of eserine were injected hypodermically.

Friday, 24*th*—One visit only was paid and eight gallons of saline solution injected. We learned that the eserine administered the previous evening had provoked great pain, but otherwise had caused no response.

Saturday, 25*th*—One visit only was paid and eight gallons of saline solution injected.

Sunday, 26*th*—Again only one visit was paid and the same quantity of saline solution injected.

Monday, 27*th*—*Morning.*—There was a history of pain all the night of Sunday and the mare was perceptibly growing thinner and weaker. The cæcum could not be felt on account of the excessive straining on the part of the patient. Owner considering destruction, to which we were not averse. Eight gallons of saline solution were injected. *Evening.*—History of pain all day. The cæcum could again be felt and although there was no sign of immediately approaching death nor any symptom of rupture, slaughter was advised.

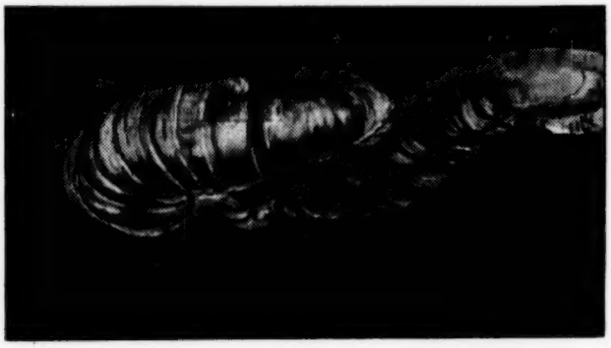
Tuesday, 28*th*—The mare was shot.

Autopsy.—This was made the same morning. Briefly the findings were those of an extensive peritonitis. The first thing noticed on making the initial opening into the abdomen was that there was no rupture. In place of the escaped ingesta found in such cases there was a flow of several gallons of inflammatory exudate, evidently septic, but not

putrid, and tinged a rose pink. The whole of the peritoneal surfaces were reddened and roughened, particularly the coats of the colon and the cæcum, while this latter organ, as we well knew would be the case, was tightly packed with a huge mass of dry ingesta. The last foot or two of the small intestine was also tightly filled, resembling a large German sausage.

Remarks. After enlarging, as I have done in former publications, on the unfortunate total abstention from water of the horse affected with impaction, I must say that during the continuance of this case I was building largely on relief arising from the large quantities of water that were periodically pumped in. Such, however, was not to occur. The fate of this mare has left me wondering now whether, in these cases, relief is ever likely to come about. In the case of a younger animal, would the issue have been different? Would the younger and unimpaired nervous system have responded to the stimulants given and allowed the continuous administration of water to exert a beneficent effect? I begin very much to doubt it.

Attached is a snapshot taken at the autopsy. The negative, however, was so thin and the resulting print so lacking in detail that it has had to be worked on with water colour until it is practically a wash drawing. It serves, however, to illustrate the cæcum greatly distended as it was with ingesta as against the comparatively empty colon.



Since the above was written I see, on page 949 of the issue of the *Veterinary Record* of November 8th, a remarkably interesting article on "Impaction of the Ileum," by D. Marshall, M.R.C.V.S., of New Zealand. It is interesting in that the author, in describing the impacted ileum, uses exactly the same simile as I have done in alluding to it here, and likens it to a German sausage. Moreover, Mr. Marshall claims that once having recognised at *post-mortem* this impacted condition of the ileum, and noted its peculiar shape, he is able to diagnose the condition by making a rectal examination, and discovering *on the right, in front of the caecum, a firm, doughy, sausage-like mass, which is the impacted ileum.* This, he says, is the diagnostic symptom; which means that Mr. Marshall now regards impaction of the ileum as a separate entity.

I mention this here for the following reason. In my own case, what I saw of the impacted ileum, which I looked upon as secondary to the impacted cæcum, convinces me that should impaction of the ileum occur independently of impaction elsewhere, then we certainly should be able, as Mr. Marshall has done, to diagnose it by rectal exploration. Should we know, as we now do, that it is to be looked for, I feel sure that in time further cases will be reported. It should be mentioned, however, that there are two factors which would militate against an exact diagnosis being arrived at in this manner. These are (1) its occurrence in an animal so large as the largest of our Shires or Clydesdales, in which it would be an almost physical impossibility to reach the seat of trouble; (2) the attendant making the exploration being of very short reach.

Nevertheless, all of us who are interested in these conditions should congratulate Mr. Marshall on his article, which is a real contribution to our slowly gathering knowledge of the intestinal troubles of the horse. As he says, however, the recognition of this condition seems chiefly valuable from a diagnostic standpoint, with a view to the more early differentiation between such a condition as this, which causes prolonged and acute abdominal pain, and volvulus.

When he refers to the fact that stimulants of bowel movement, including the use of the alkaloid cathartics, has grown to be widely used as routine treatment and on that account makes the statement that accurate diagnosis is less important, I cannot say that I quite agree with him. I grant him that the mere fact of diagnosing it makes no difference to the issue of the case, provided the treatment is on right lines. Accurate diagnosis, however, I should never in any way, no matter in how slight a degree, hold as unimportant. Although it may be opposed to the opinion of many, I still believe that the day will come when an increasing knowledge of the diagnosis of these conditions will lead to an advance in abdominal operations for this and allied troubles in the horse, and Mr. Marshall's contribution I regard as one more step along the road.

The last horse-drawn "Black Maria" was taken off the London streets on November 18th. The first "Black Maria" ran in 1838, and for the past 30 years Messrs. Tilling have had the contract for supplying horses.

In view of the interest which is now being shown in the desirability of feeding hard biscuit foods to dogs and the publicity being given to such matters, Messrs. Wright and Co., Liverpool, are issuing a booklet, "Dogs—and Hard Tack," with a "Foreword" by Major Harding Cox.

The booklet, in which a good case is made for the feeding of the firm's speciality biscuits, will be sent post free to any readers of the *Veterinary Record* upon a receipt by Messrs. Wright of a request to that effect.

Central Veterinary Society.

Fifty-First Annual Dinner.

A Memorable Social Function.

Fellows, guests and lady visitors to the number of 117 supported the President of the Central Veterinary Society, Major P. J. Simpson, at the fifty-first annual dinner, which was held at the Trocadero Restaurant, on Thursday, November 6th, 1924.

The two chief innovations made on this occasion—the change of venue and the emphasising of the social aspect of the function—were a pronounced success, as was proved by the rapidity with which the evening passed despite, or by reason of, the limited time devoted to speech-making.

The evening commenced, in accordance with custom, with the President's reception, the element of sociability being considerably enhanced at the subsequent repast by the seating of the company at small *tables d'intime*.

The success of the dinner was contributed to so greatly by the enlarged musical programme that mention may be made thus early of the pleasure afforded the company by the delightful soprano singing of Mrs. Brakspeare, a personal friend of the President's, while the efforts of the following professional artistes were also much appreciated:—Miss Lilian Sterling (contralto), Mr. Charles Jackson (baritone), and Messrs. A. E. Nicholls and A. H. Howe, who were very effective in humorous harmony.

Following the honouring of the Loyal Toasts, Sir D'Arcy Power submitted "The Central Veterinary Society." It was an especial pleasure to him to propose that toast, because it gave him the opportunity of emphasising once more the great friendship which had always existed between the human and veterinary sides of the great profession of medicine. That relationship had existed from the earliest times—from those of St. Bel, and down through the days of that great pioneer of veterinary work and surgery, John Hunter, through the surgeons connected with him, up to the days when Sir John Bland Sutton was one of their great teachers.

He brought with him that evening a message—Sir John had told him that he would have liked to have been with them, but he was dining with another branch of the veterinary profession, the skinners. (Laughter.) He replied that he thought, nevertheless, that Sir John should have accompanied him to dine with the lions. (Laughter.)

That Society was founded half a century ago, and it was founded to take in the élite of the profession. For some reason they had then thought it should be called the Pathological Society, but eventually they adopted the wider term of the Central Veterinary Society.

During the last fifty years the whole of the science of veterinary medicine and surgery had been revolutionised, until they had reached the science of comparative medicine—a much wider term, because it embraced that aspect of medical activity which he represented, as well as their own.

With those few words he gave them the health and wealth, long prosperity and life of the Central Veterinary Society. (Cheers.)

The President, in reply, thanked Sir D'Arcy Power, on behalf of the Fellows of the Society, for the terms in which he had proposed the toast. The history of the Central Veterinary Society had been gone into at previous functions so many times that he did not propose to make it the subject of a long speech on an occasion which he looked upon as the social event of the year, when they met together for friendship's sake more than for the purpose of thinking of things historical or scientific.

It was, however, very pleasant to them all to know that that Society had progressed, that it was continuing to do so, and that there were no signs of retrogression. It was really very flattering to the officers of the Society to know that the assembly there that night had exceeded the highest number that had ever previously attended one of those gatherings. (Cheers.)

The work of the Society was always, as they knew, chiefly incumbent upon one or two, the one or two being their executive officers, the treasurer and the secretary. No society could progress unless it had competent executive officers; that Society was well served in that respect. (Hear, hear.) In Mr. Macdonald, as secretary, they had a very energetic, a very capable and a very conscientious man, who carried out his duties in the most able and thoroughgoing manner. The same remarks applied to his good friend Mr. Stroud; they had known each other for many years, and he was sure that the Society, too, needed no reminder of the many long years of excellent service which their treasurer had rendered them. They had to look forward to the future; he hoped that the coming session would be as brilliant as the past had been. (Cheers.) He trusted that there would be plenty of good papers, containing much food for thought, and that they would arouse ample and instructive discussion and criticism of the helpful as opposed to the carping kind. He also hoped they would encourage in attendance not only the younger members of the profession, but those as yet unqualified, to whom they would, after qualifying, look for recruitment, and it would be his pleasure to see some of the final year students at College come to their meetings. By that means, he thought, the Society would be sowing the seeds of patriotism and loyalty, so that these men might become good members of the Central Veterinary Society and of the National Veterinary Medical Association, of which that Society was one of the divisions. (Hear, hear.) Short speeches were decided upon by the Dinner Committee, for which he was truly thankful (laughter), but before closing he desired to say that he had approached that dinner with very great misgivings, one untoward thing having happened after another—in the case of the guests it was almost a case of the ten little nigger boys (laughter); he had,

indeed, had many disappointments, but when he saw the large number present all those troubles were forgotten and he thanked them all very much indeed for supporting the chair so splendidly as they had done that evening. (Cheers.)

PRESENTATION OF THE VICTORY MEDAL.

The PRESIDENT: We now come to what, I think, is the most pleasant part of the evening—I am sure it is to me, and it must be to you also. It is the presentation of the Victory Medal. Most of you are aware of the origin of the Victory Medal, and how and why it is awarded. To those who do not know, I may say that the Society decided that, to commemorate the victory of England and her Allies in the Great War, this medal should be struck. The rules regulating the giving of this medal are somewhat wide, but it is awarded to the member of this Society who has performed the greatest work for the Society and for the profession in general.

This evening it has been allotted to me—a very great privilege—to present this medal to a member of this Society, known to all and beloved by all; I speak of Mr. J. W. McIntosh. (Cheers.) To enumerate all the qualifications that Mr. McIntosh has which render him deserving of the receipt of this medal would be far too long an undertaking to-night, but I will endeavour as briefly as possible to mention just a few of these qualifications. In order to do so, one must almost begin to classify these qualifications as to those connected with his public career and those which have to do with his career as a professional man. Mr. McIntosh is one of those forceful Scotsmen who make their presence felt wherever they go, not in an objectionable way, but in a quiet, unobtrusive and none the less effective way. Mr. McIntosh was a member, in his younger days, of a Scottish yeomanry regiment. If he did not reach the rank of Colonel Commandant he must, at any rate, have achieved the no less important position in the regiment of sergeant-major. (Laughter.) Soon after he settled down in practice at Dalkeith, he became a member of the Council and he soon took up the public health side of Council and Municipal work. He was one of the instigators of the establishment of bye-laws and reforms connected with dairies and cowsheds within the area governed by that Council, when many a man would have been frightened to do such a thing for fear of offending his clients in the neighbourhood. That goes to show that Mr. McIntosh, at an early date in his career, was a pioneer in public health work. (Cheers.) So good was his work on the Council that, when he left Dalkeith, he was presented with a handsome token of the esteem in which he was held by the mayor and corporation and the townspeople.

With regard to his professional activities, they are legion. He qualified in 1893 at the "Dick" College and for fourteen years he practised in Dalkeith, during which time he held numerous public health appointments under the Public Health and Contagious Diseases Act of Scotland. He became one of the governors of the "Dick" College and a life member of the Highland Agricultural Society. In 1908 he came to London and took up his duties in connection with the professional management of one of the largest commercial studs of horses in England. He is now one of the chief authorities on all matters connected with horse transport.

Since he came to London, Mr. McIntosh has closely identified himself with the work of the profession in the South. He has been a member of many societies. He is, at the moment, President of the society in which I happen to hold the position of secretary, the Royal Counties Society. He is a member of this Society, of the Southern Counties, and of the South Eastern; he is also a member of Council and many committees of the N.V.M.A. The same is also true of the R.C.V.S. I happen to be running a good deal in harness with him in the performance of some of these duties and I can say that Mr. McIntosh puts forward his views clearly and emphatically, and he has a way of carrying his point and getting things done. (Hear, hear.) There is no one who has had any experience of Council work, either of the "National," or the R.C.V.S., who is not well aware of the vast amount of work which goes on behind the scenes in committee—there are few outside who realise that a committee may have to meet twenty or more times to thrash out a subject that comes up before the Council. Mr. McIntosh is one of the foremost of the people who attend these small meetings so that the Council may have a clear way in front of them. One of the big things that Mr. McIntosh has done has been to take up the cudgels on behalf of veterinary inspectors and those holding colonial appointments, and I think I may say that his efforts have not been in vain, because there is every evidence that, in consequence of the good work that he and his colleagues have done in this connection for the N.V.M.A., that body is being listened to by departmental bodies and others.

Finally, you all know what a good friend to us all is Mr. McIntosh. (Cheers.) With these totally inadequate remarks I will ask Mr. McIntosh to come forward and accept this medal.

(To be continued.)

NOTES AND NEWS.

Diary of Events.

Nov. 29th—R.C.V.S. Written Examination for D.V.S.M.

Dec. 1st—4th—Oral and Practical D.V.S.M. Examination Edinburgh and London.

Dec. 8th—R.C.V.S. Written Examinations—Membership Diploma.

General Meeting of the Association for the purpose of voluntary liquidation.

Dec. 11th—R.C.V.S. Oral Examinations commence in Dublin.

Dec. 23rd—Confirmatory General Meeting of the Association.

1925.

Jan. 1st—Nominations for election to Council R.C.V.S.

Jan. 7th—Foreign Voting Papers R.C.V.S. issued.

Jan. 7th—R.C.V.S. Examination Committee, 2 p.m.

Jan. 8th—R.C.V.S. Committee Meetings, 11 a.m. to 1 p.m., 2 p.m. to 5 p.m.
Victoria Veterinary Benevolent Fund Council, 5 p.m.
Jan. 9th—Publication Committee R.C.V.S., 11 a.m.
Finance Committee R.C.V.S., 12 noon.
R.C.V.S. Council Meeting, 2 p.m.
R.C.V.S. Special Council Meeting, 3-30 p.m.
Jan 22nd—R.C.V.S. Special Council Meeting (probable date).

We have received intimation of the death of Mr. W. P. Stableforth, Honorary Secretary of the Western Counties Division, which occurred at his home, Honiton, Devon, on Saturday, 22nd inst. This information will be received with very great regret by all those who are not already aware of it. Mr. Stableforth is very well-known in the profession, and he has many friends who remember the excellent work he did for the Association as Local Secretary at the Bath Congress two years ago.

The Laurencekirk Town Council have by a unanimous vote elected Mr. H. W. Robson, M.R.C.V.S., Provost and Chief Magistrate of the borough. The new Provost was co-opted as a member of the Town Council in 1917 and since then he has contested two elections and on each occasion headed the poll by a large majority.

R.C.V.S. Diploma in Tropical Medicine.

At a meeting of the Panel of Examiners in Tropical Medicine, held at the College, 10 Red Lion Square, W.C.1., on Thursday, November 20th, the following candidate was successful in obtaining the Diploma :—

Captain Colin Davenport, O.B.E., R.A.V.C.

Title of Thesis :—Cameline Surra : *Some observations in connection with its Course, Diagnosis and Treatment.*

Examiners :—F. S. H. Baldrey and A. Leslie Sheather.

Chairman :—Mr. S. H. Slocock (President R.C.V.S.).

Royal College of Veterinary Surgeons.

Obituary.

Kelly, Major Edward, M.R.C.V.S., late A.V.D., died at St. Anne's, Blarney, C., Cork, on November 12th, 1924.

Major Kelly, who graduated on May 26th, 1852, at the Royal Veterinary College, Camden Town, was the " Father of the Profession," having been a member of the R.C.V.S., for over 72 years.

Foot-and-Mouth Disease in Kent.

An outbreak of foot-and-mouth disease at Chatham was declared at the end of last week by the Ministry of Agriculture, and an area of Kent, comprising Rochester and the boroughs of Chatham, Gillingham, Maidstone, Gravesend, and Queenborough, as well as a number of petty sessional divisions, was notified in the *London Gazette* as an infected area in which the movement of cattle is subject to regulations.

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CORRESPONDENCE.

Parasitology.

To the Editor of the Veterinary Record.

Sir,—I am not one of those who believe that knowledge of a theoretical kind in connection with the above is badly taught in our Colleges and Schools. I have seen plenty of the students' notes from two or three of the places of instruction. What I should, however, like to know is whether in any of the teaching establishments there is a fairly complete collection of examples of the parasites encountered by us in connection with all our animals (horses, cows, pigs, sheep, dogs, cats, fish, fowl, etc.). It is no use knowing all about the parsaites if we cannot recognize them when we see them because, if our clients ask us about them, we can only plead relationship to Mr. Kremlin, who " was distinguished for ignorance, for he only had one idea and that was wrong."—Yours truly, G. Mayall, M.R.C.V.S.

Bolton.

The Veterinary Colleges in Scotland.

To the Editor of the Veterinary Record.

Sir,—Your article of the 22nd November, states the case for the Royal (Dick) very well indeed, but I feel sure that I can supply arguments which will modify your own attitude considerably.

In a matter of this importance, I naturally have to submit any extended statement to the Governors of Glasgow Veterinary College, before forwarding it for publication, and I hope that you will suspend definite judgment until you have heard our side.—Yours faithfully,

A. W. Whitehouse, *Principal.*

Glasgow Veterinary College (Incorporated), 83 Buccleuch Street, Glasgow,
24th November, 1924.

The Editor acknowledges the receipt of the following :—Communications from Captain W. Wallace Kerr, Mr. W. Shannon, Major General Sir F. Smith.

A letter from Professor B. Gorton is unavoidably held over through lack of space.

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Edited by G. H. Wooldridge, F.R.C.V.S., M.R.I.A., F.Z.S.

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